A MOBILE DEVICE-BASED INTERVENTION TO REDUCE THE INFLUENCE OF SMOKING CUES AMONG AFRICAN AMERICAN CIGARETTE SMOKERS

by

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ABSTRACT

A Mobile Device-based Intervention to Reduce the Influence of Smoking Cues among African American Cigarette Smokers

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Introduction: African American cigarette smokers have higher rates of lung cancer and lower rates of smoking cessation compared to Whites. African American smokers also live in communities that have a disproportionately high number of tobacco cues and advertisements. Exposure to smoking cues may promote smoking and undermine cessation attempts. While it is difficult to modify the number of smoking cues in the environment, it may be possible to reduce attention to those cues ("attentional bias"). This procedure is termed Attentional Retraining (AR), which trains smokers to attend away from smoking cues. AR may reduce exposure to smoking cues, and therefore reduce craving and smoking. The current study examined the efficacy of AR administered on a mobile-device in reducing attentional bias, craving, and smoking.

Method: Non-treatment seeking African American cigarette smokers (N = 64) were randomly assigned to an AR or Control training condition. Participants were given a mobile-device for 2 weeks, which prompted them to complete up to three AR (or control) trainings per day. Participants completed assessments of attentional bias, craving, and smoking both in the lab and in the field (one time per day).

Results: Overall, participants initiated 2,419 trainings and assessment. Participants in the AR and Control conditions completed an average of 29.07 AR (*SD* = 12.48) and 30.61 control training tasks (*SD* = 13.07), respectively. AR reduced attentional bias assessed in the laboratory, F (1,126) = 9.20, p = .003, and field, F (1, 374) = 6.18, p = .01. This effect generalized to new stimuli, but did not generalize to new tasks. AR did not significantly reduce craving but did reduce smoking assessed on the mobile device. Smoking declined over days in the AR group, F (1, 26) = 10.95, p = .003, but not in the Control group, F (1, 27) = 0.02, p = .89. AR did not reduce biochemical measures of smoke exposure.

Discussion: Two weeks of AR administered on a mobile device reduced attentional bias in African American smokers. AR also reduced reported smoking over time. Further research is necessary to clarify the mechanisms underlying the effect of AR on smoking.

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KEY TERMS

AR = Attentional Retraining (in this study AR was delivered by Modified Visual Probe task in which probes only replace neutral stimuli)

Assessment = a Personal Digital Assistant (PDA) field assessment (delivered during EMA) that includes the presentation of the standard Visual Probe (VP) task (in addition to measures of cued craving, non-cued craving, exposure, and smoking) (AR or Control Trainings are not delivered at Assessments)

Attentional bias (aka positive attentional bias, or simply "bias") = tendency to attend to smoking stimuli (faster responses on probes that replace smoking stimuli)

Avoidance (aka negative attentional bias) = tendency to attend away from smoking cues (faster responses on probes that replace neutral stimuli)

Control Trainings = VP task in which the probes replaced smoking and neutral pictures with equal frequency (received by Control group but not by AR group)

Cued Craving = craving assessed after a 1-second presentation of a picture containing neutral and smoking cues

EMA = Ecological Momentary Assessment (methodology in which participants responses are collected in real time in the field)

LMM = Linear Mixed Models (a method to analyze EMA data in which each subject has multiple datapoints)

Negative Attentional Bias = Avoidance (faster responses to probes that replace neutral stimuli)

Non-Cued Craving = craving assessed without the prior presentation of a picture

Participant-initiated assessment = a PDA field assessment (Training or Assessment) initiated by the participant

PDA = personal digital assistant

PDA field assessments = Trainings/Assessments completed in the field; ~75% of PDA field assessments were Trainings (AR vs. Control) and ~25% were Assessments (as defined above)

RA = Random Assessment (aka PDA-initiated assessment, an assessment initiated by the PDA). Trainings & Assessments are both delivered at RAs

Standard VP task = "Assessment" version of task in which the probes replace smoking and neutral pictures with equal frequency

Trainings = A PDA field assessment that includes the presentation of AR or Control training task (in addition to measures of cued craving, non-cued craving, exposure, and smoking)

VP task = visual probe task (task in which participant have to make a decision about to location of visual probes, in this study dots)

CHAPTER 1: Introduction

African American cigarette smokers have higher rates of lung cancer and lower rates of cigarette smoking cessation than White smokers (208; 222). Current smoking cessation treatments have had limited effectiveness among African Americans (174). Therefore, there is a pressing need to develop new smoking cessation interventions for African American smokers. African American smokers also live in communities that have a disproportionately high number of tobacco cues and advertisements in contrast to Whites (181). Exposure to smoking cues may promote smoking and undermine cessation attempts (26). While it is difficult to modify the number of smoking cues in the environment, it may be possible to reduce attention to those cues ("attentional bias"). This procedure is termed Attentional retraining (AR), which trains smokers to attend away from smoking cues (11). AR may reduce exposure to smoking cues, and therefore reduce craving and smoking. The current doctoral dissertation research examined the efficacy of two weeks of AR administered on a mobile-device in a sample of 64 African American smokers. The primary aims were to examine the ability of AR to reduce attentional bias, craving, and smoking in the laboratory and field (i.e., in the participants daily environment). The following rationale for the current study provides a roadmap of the literature review that follows.

Rationale for Current Study

The rationale for an AR intervention for African American smokers is as follows: (1) Tobacco use is the leading cause of death and disease (2) Many African American smokers are motivated to quit but most quit attempts end in failure; (3) Traditional treatments have had limited success in African American smokers; (4) African

Americans live in cue-rich environments and therefore are more exposed to smoking cues; (5) Exposure to smoking cues is associated with craving and use/relapse. In the current study, AR delivered on a mobile device was used to try to reduce the influence of smoking cues. If AR reduces attentional bias, African American smokers may be better able to navigate their cue-rich environments. Long-term, this intervention could be paired with other efficacious interventions. Also, AR is inexpensive and may be implemented on mobile devices such as smartphones which would make mass dissemination feasible.

Tobacco Use and Health

Tobacco use is the leading cause of preventable disease and death in the United States (4). Approximately 480,000 individuals die from tobacco-related illnesses per year (4). The use of cigarettes in the U.S. is particularly concerning because in 2013 the smoking rate among adults 18 and older was 17.8% (4). These rates are alarming because tobacco use has been linked to multiple cancers, cardiovascular disease, pulmonary disease, and the exacerbation of other illnesses (74; 195). Given that millions of Americans still smoke cigarettes and tobacco use costs the U.S. 289 billion dollars in medical costs and productivity per year, expanded efforts are needed to reduce cigarette smoking (4).

Tobacco Use and Health Disparities

The health consequences of tobacco use are far reaching and affect all demographics (32). However, there are subgroups within the U.S. population that are disproportionately impacted, such as racial/ethnic minorities (57). It is clear that racial/ethnic minorities are disproportionately affected by the health consequences of tobacco, suggesting a significant health disparity. Health disparities are defined as

"…differences in the incidence, prevalence, mortality, and burden of disease and other health consequences among a specific population (2)." These differences are typically associated with some level of inequality (30).

Health inequity is an additional term used to describe differences in health among subgroups (236). Health inequity takes into account differences that are avoidable and caused by injustice (24). The impact of injustice and systematic inequalities on health has become a major focus among researchers (237). As a result the term health disparity is also used to describe differences in health as a result of injustice (24). Conceptually, one can view health inequity as an independent variable and health disparity as a dependent variable, and health inequities can be said to cause health disparities. However, in the literature the terms tend to be used interchangeably. Therefore, in the current text both health inequity and health disparity are used interchangeably.

Social Determinants of Health

Health Disparities among African American smokers must be considered in the context of the broader literature on the social determinants of health. The social determinants of health refer to nonmedical factors that influence health including the conditions where people are born, grow, live, work, and age (96; 237). In recent years there has been a growing interest in the social determinants of health because they lead to health inequity that is avoidable and unnecessary (237). Braveman and colleagues (25) provide a powerful conceptualization of how health inequities develop. In their conceptualization, economic opportunities and social resources are upstream (i.e., controlled by policy) social determinants of health that influence the conditions that people live and work. The conditions in which people live and work influence medical

care and health behavior which are considered downstream (i.e., a more proximate cause of illness) determinants of health. Braveman and colleagues (25) note that the upstream social determinants of health (economic opportunities and social resources) are fundamental in health inequity and inequality. Solar and Irwin (211) present a similar conceptualization and emphasize the influence of government policies on upstream social determinants of health (e.g., the economic and social resources).

Research on health disparities in the U.S. has focused largely on differences by race. Race in the U.S. is correlated with the social determinants of health described above, including the availability of social and economic opportunities (238). However, the approach of focusing on race has been critiqued because there is evidence that socioeconomic status is more directly related to health disparities (97). Moreover, there is a growing interest in evaluating the factors that contribute to racial differences in health. Residential segregation is an example of a factor that contributes to health disparities among African Americans (238). Although no longer supported by legal policies, residential segregation is still pervasive in the U.S. Moreover, the U.S.'s history of residential segregation contributes to differences in educational and economic opportunity. Partly as a result of racial segregation, African Americans are disproportionately represented among the low socioeconomic status, which is linked to poor health outcomes (213).

The effect of residential segregation on health is empirically supported (50; 121). LaVeist (121) investigated the health outcomes in an integrated neighborhood in Baltimore, Maryland. He found that the racial differences in health outcomes were

similar among African Americans and Whites who lived in the same neighborhood. Stated another way it *is place not race* that contributes to health inequity (121).

The above review of social determinants of health highlights the importance of a macro-level perspective of health disparities. This view indicates that upstream factors such as economic resources influence health outcomes and cause disparities in health. Interventions targeted at improving access to economic resources are important for reducing health disparities. Although, this doctoral dissertation is focused on cigarette smoking, a downstream factor, it is essential that parallel efforts are made to improve the policies that influence economic opportunity.

Finally, research in race-related health disparities should be conducted with caution because it requires categorizing individuals into groups based on the social construct of race. The categorization of individuals by race has a negative history in the U.S., particularly as it relates to prejudice and discrimination. Hence, a great deal of controversy exists among researchers and laypeople regarding how (or if) individuals should be categorized (101). For the purpose of this paper, race was used to distinguish individuals who self-identify with a specific cultural group and ancestral geographical location. The terms used to describe races are aligned with those used by the Office of Management and Budget guidelines on terminology for race and ethnicity (161). The category African American was used to refer to all Americans of African descent (including Caribbean Americans and immigrants from Africa).

Health Disparities among African American Smokers

Historically, African Americans have had the highest rates of tobacco-related morbidity (223). Recent data reveal that the death rate and incidence rates of lung cancer

are higher among African American men compared to White men (i.e., smokers and nonsmokers). Data from 2004 to 2008 revealed that the incidence of lung cancer was 22% greater among African American men when compared to White men and similar among African American women and White women (32; 208). Between-race differences may be due to a variety of factors including accessibility to treatment, physician advice, low rates of nicotine replacement therapy among African Americans, and economic disparities (57; 82; 208).

For example, there is evidence that African Americans have lower rates of nicotine replacement therapy use than White smokers in an equal access health care system (72). Although the mechanisms of this difference are not well-understood, there is evidence that medical mistrust (developed from the exploitation of African Americans in medical research) may prevent African American smokers from taking medical advice (122). Although the cost of nicotine replacement therapy may represent a barrier to nicotine replacement therapy use among African Americans, there is currently little empirical evidence to support this hypothesis (72). As noted above, there is evidence that underutilization of nicotine replacement therapy occurs among African Americans even when cost is not a barrier (72). This finding suggests that other factors including medical mistrust, physician advice, and the marketing of nicotine replacement therapy (242) may be important.

In addition to suffering from greater tobacco-related morbidity, African American cigarette smokers also have greater difficulty quitting cigarette smoking when compared to White smokers (222). Differences in smoking cessation are expanded upon later in the text are noted here to highlight the need for additional research on African American

smokers. Furthermore, it is evident that African Americans have been understudied in the tobacco literature (58; 153). Specifically, there is limited knowledge on patterns of use, cessation, and withdrawal among African American smokers.

Cigarette smoking among African Americans is expanded upon later in the text to further support the need for a novel and culturally targeted smoking intervention. First, it is necessary to review nicotine addiction and smoking cessation treatment in general. This background is necessary to fully understand and treat African Americans who smoke.

Nicotine Addiction

Reducing cigarette consumption in the U.S. is a challenging task because of the addictive properties of nicotine, the primary psychoactive ingredient in tobacco (81; 223; 225). Nicotine addiction is a chronic, relapsing brain disorder characterized by compulsive use, despite harmful consequences, and the appearance of withdrawal symptoms upon cessation (125; 157). Nicotine addiction results from the interaction of environmental factors, genetics, learned and conditioned factors, and pharmacology (18). This paper reviews the neurobiological, learning, and environmental factors that contribute to addiction. The terms "addiction" and "dependence" are often used synonymously in tobacco research (18; 235). In this paper, the terms are used interchangeably because they both characterize a loss of control over drug-taking behavior.

Neurobiology of Nicotine Addiction

The pharmacological actions of nicotine have been well-studied and are necessary to fully understand nicotine addiction. Nicotine is an alkaloid that acts on nicotinic

acetylcholine receptors (nAChRs) distributed throughout the central nervous system and the peripheral nervous system (33; 139). In the context of addiction, nicotine is an agonist that binds to the α4β2 subunit of the nAChR complex in the Ventral Tegmental Area (VTA), an area of the midbrain associated with the motivational effects of drugs of abuse (148). After nicotine binds to nAChR receptors in the VTA, the ascending neurons of the VTA project to the nucleus accumbens, corpus striatum, and the prefrontal cortex, which causes dopamine to be released in these area (80). Nicotine modulates the release of other neurotransmitters such as norepinephrine, vasopressin, acetylcholine, serotonin, and β-endorphin. These transmitters are involved with the pleasurable psychoactive effects of nicotine such as arousal, cognitive enhancement, mood modulation, and appetite suppression (22). In chronic smokers, nicotine reduces the activity of Monoamine Oxidase A and B, which are enzymes that break down dopamine and norepinephrine (19). Reduced enzyme activity results in even more dopamine and norepinephrine in the synapses. The elevation of neurotransmitters contributes to the development of nicotine addiction. Repeated nicotine administration results in neuroadaptations such as the upregulation of nAChRs in response to desensitized receptors (15).

Nicotine Withdrawal

An additional feature of nicotine addiction is nicotine (1; 86). During periods of abstinence or sleep, many smokers experience a withdrawal syndrome. This syndrome occurs during and after nicotine is eliminated from the body. Smokers experience symptoms that are opposite to what they experienced when using nicotine. For instance, abstinence from tobacco has been reported to produce subjective, physiological, and cognitive changes (86; 92; 94). Subjective symptoms include negative affect, irritability,

difficulty concentrating, craving, anxiety, and dysphoria (7) Hughes (92; 93). The primary physiological/somatic symptoms are bradycardia, decreased blood pressure, increased appetite, and gastrointestinal discomfort (86;107). Abstinence from tobacco also affects objective cognitive performance as documented by decrements in performance on tasks of sustained attention (93; 127).

There are also abstinence-induced changes in brain electrical activity as detected by electroencephalogram (172). The electroencephalogram (EEG) is a non-invasive tool that can be used to measure abstinence-induced changes in the central nervous system. Previous research has demonstrated EEG changes following overnight abstinence (89; 112; 172). For example, abstinent smokers exhibit increases in theta power (associated with diminished arousal) and decreases in alpha power (associated with drowsiness) compared to non-abstinent smokers (89; 172)

Nicotine's short half-life (approximately 2 hours) results in the experience of withdrawal symptoms and craving that develop within several hours of the last cigarette ((196). The nicotine withdrawal syndrome tends to peak within two to three days and most symptoms return to baseline within 10 days (205). However, there is evidence that craving and increased hunger persist for up to 6 months or longer (80; 93).

Theories of Nicotine Addiction

In summary, cigarette smoking is addictive due to the addictive properties of nicotine. Related to the previous discussion, there are three primary psychological explanations for nicotine addiction: positive reinforcement, negative reinforcement, and the conditioning of stimuli associated with use. These constructs are important because

interventions have been developed to target these various aspects of nicotine addiction. A review of this literature is presented below.

Positive Reinforcement

Learning principles have been utilized to characterize the development and maintenance of nicotine addiction. One view of nicotine addiction is that tobacco use is maintained by the positive effects of nicotine such a s arousal, relaxation, and improved mood (217; 239; 240). This view is referred to as a positive reinforcement view of addiction because the drug (the stimulus) is immediately followed by a reward (e.g., arousal) (15; 115). This view is supported by evidence in which animals and humans self-administer nicotine to obtain the positive reinforcing effects (88). However, current theories suggest that positive reinforcement is not sufficient to cause addiction (188). Much of this criticism comes from continued self-administration in the absence of pleasurable effects. In addition, research indicates that euphoria received from nicotine is modest (187). This evidence suggests that the euphoria from smoking is not substantial enough to maintain addiction. Self-administration in the absence of pleasure and modest euphoria obtained from nicotine suggests that alternative explanations of nicotine addiction should be examined.

Negative Reinforcement

An additional explanation for addiction is the negative reinforcement view. Historically, this view has generated a lot of support and research. In this model, drug use is maintained because it relieves withdrawal symptoms (113; 212). Proponents of this view argue that drug use is not maintained by the positive effects that it produces but by the ability of drugs to relieve a negative state (239). However, research suggests that

negative reinforcement may not fully explain addiction. This criticism is in part due to evidence in human and animal research in which relapse occurs after withdrawal symptoms have subsided (241).

Opponent-Process Model

The opponent-process model is a third model of drug addiction (114; 212). This model suggests that drugs activate a dose dependent positive hedonic state that automatically triggers a negative state. The activation of the negative state is believed to restore homeostasis. Overtime, the magnitude of the negative state increases while the positive, hedonic state diminishes. This model proposes that the negative state maintains drug use (212). The opponent-process model is also limited by its inability to explain relapse that occurs after withdrawal symptoms have subsided (188).

Incentive Sensitization Theory

A third explanation for addiction that is particularly pertinent to the current study is the Incentive Sensitization Theory (IST) (188). Robinson and Berridge state that addictive drugs alter nucleus accumbens-related brain systems such that neural circuits become hypersensitive to drug-related stimuli, thereby assigning salience to drug cues. As such, drug use is maintained by the ability of the conditioned stimuli (i.e., drug cues) to trigger motivation for drugs (188). These associations develop through the processes of classical conditioning. Moreover, drug cues become so salient that they cause drugs to be wanted, independent of any pleasure they yield (189). As described in more detail later, this theory is particularly relevant to the current proposal because it suggests that drug cues should grab the attention of drug dependent individuals (see "attentional and addiction"). Moreover, as will be described in detail later, attention to drug cues (or

"attentional bias" to drug cues) will be the target of the proposed intervention. First, a brief review of the smoking cessation literature is warranted.

Smoking Cessation

Many adults are motivated to quit smoking but most quit attempts end in failure (32). For instance, the National Health Interview Survey (NHIS) indicated that in 2010, 68% of adult smokers reported wanting to quit smoking (32). Among those interested in quitting, 58.8% made a quit attempt (they maintained abstinence for more than 1 day). However, only 6.2% were successful at maintaining abstinence for six months (32). Many smokers interested in quitting utilize treatments such as pharmacotherapy and/or counseling. The NHIS survey also found that 30% of smokers who tried to quit in the past year utilized a pharmacological treatment and 5.9% utilized a behavioral/counseling intervention (32)

There has been extensive research examining the efficacy of pharmacological treatments and psychological interventions. A brief review of the most relevant data is presented below. The following review also highlights the variety of evidence-based pharmacological and behavioral treatments available to smokers including African American smokers. As discussed in the section on 'Smoking cessation among African American Smokers,' despite the availability of these treatments few African American smokers are able to maintain abstinence. Furthermore, prior to understanding the smoking cessation treatment tested in the current doctoral dissertation a review of current evidence-based treatments for smoking are necessary.

Pharmacological Treatments

There are several pharmacological treatment options available to smokers. First-line treatments (as recommended by the Department of Health and Human Services) include nicotine replacement therapies, varenicline, and bupropion (159;76). These treatments were approved by the Food and Drug Administration (FDA) as first-line treatments because they reliably increase smoking abstinence and have few adverse events (159).

Nicotine Replacement Therapies

Nicotine replacement therapies target the withdrawal and craving symptoms experienced by smokers during abstinence (209). This treatment option provides smokers nicotine that (partially) replaces what they normally received from tobacco (18). There are seven formulations of nicotine replacement therapy including nicotine inhalants, nicotine gum, nicotine tablets, nicotine lozenges, nicotine transdermal patches, nicotine sublingual tablets, and nicotine nasal spray. The type of nicotine replacement therapy that is selected is driven by patient preference. Patients typically select the type based on the route of administration, perceived adverse effects, advertising, and price (159). There are some modest differences in the efficacy of different nicotine replacement therapy formulations. However, research suggests that the difference is explained by adherence (159).

The efficacy of nicotine replacement therapies has been investigated in several randomized clinical trials (54; 209). For example, Eisenberg and colleagues (51) conducted a meta-analysis of 69 randomized clinical trials. This analysis revealed that nicotine replacement therapy roughly doubles a smoker's chance of cessation (when

compared to placebo); nicotine gum (*OR* = 1.71, 95% CrI = 1.35 - 2.21), nicotine nasal spray (*OR* = 2.37, 95% CrI = 1.57- 3.60), transdermal nicotine (*OR* = 2.07, 95% CrI = 1.69 - 2.62), and nicotine tablet (*OR* = 2.06, 95% CrI = 1.12 - 5.13) (CrI = Credible Interval, a parameter used in Bayesian statistics).

Bupropion (Wellbutrin)

Bupropion is a non-nicotine agent, better known for its use as an anti-depressant (210). The mechanisms of bupropion as an aid in smoking cessation are not well-understood. However, it is known that bupropion is a weak nicotine antagonist that blocks norepinephrine and dopamine reuptake (159). Additionally, bupropion is a nicotinic antagonist that decreases craving and withdrawal (220). Meta-analyses have revealed that bupropion substantially increases the odds of cessation (51; 215). For example, Eisenberg and colleagues (51) found that bupropion doubled the odds of cessation, (*OR* = 2.07, 95% CrI = 1.73 to 2.55) when compared to placebo.

Varenicline (Chantix)

Varenicline is also an FDA-approved agent for smoking cessation (159; 176). Varenicline is a partial agonist and antagonist for the neuronal nicotinic acetylcholine receptor subtype $\alpha_4\beta_2$ (146). Varenicline provides relief from withdrawal and craving by stimulating the release of low levels (30% to 60% less than nicotine) of dopamine (36). Also, Varenicline competitively binds to the nicotinic acetylcholine receptor subtype $\alpha_4\beta_2$ and $\alpha 7$ (159;59). This action inhibits dopaminergic activation from smoking (i.e., it prevents the reinforcing effects of nicotine). Studies also suggest that Varenicline significantly improves the odds of cessation (102; 164). For example, Eisenberg and colleagues (2008) conducted a meta-analysis and reported that Varenicline more than

doubled the odds of cessation (*OR* = 2.41, 95% CrI = 1.91 - 3.12) when compared to placebo.

In summary, there is a great deal of evidence to support the use of pharmacological treatments to help smokers achieve abstinence. However, there is certainly room to improve smoking cessation outcomes. Treatments that target psychological aspects of dependence can serve as an adjunctive to medications. Moreover, the Department of Health and Human Services guidelines for treating nicotine addiction recommend that smokers utilize both psychological and pharmacological treatments (68).

Psychological Treatments

In addition to pharmacotherapy, smokers can choose from a variety of psychological treatments such as cognitive behavioral therapy (CBT) and motivational interviewing (MI). Psychological treatments are typically provided in three primary modalities: individual counseling, group counseling, and counseling through smoking quit-lines. Many of the psychological treatments target constructs such as motivation, cognition, and behavior. For instance, in motivational interviewing a counselor may highlight a smoker's discrepancy about the benefits and consequences of smoking (119). This awareness and discomfort may provide the motivation to resolve the discrepancy.

Individual Counseling

Psychological treatments for smoking cessation have been extensively investigated in randomized controlled trials (155). Cochrane reviews and numerous meta-analyses have found psychological treatments to be superior to control interventions (e.g., minimal contact and varied treatment intensities). For example, individual counseling

interventions delivered by specialist counselors (i.e., not routine care from a physician or nurse) were investigated in a Cochrane review (120). The interventions included face-to-face individual counseling for participants not utilizing pharmacotherapy. The types of therapeutic approaches in the studies varied. The interventions were compared to minimal contact interventions, different approaches, and different intensities of counseling. The meta-analysis of 30 randomized controlled trials revealed that individual counseling was better than a control intervention (risk of smoking cessation at long term follow up, *RR* = 1.39, 95% CI = 1.24 - 1.57).

Group Counseling

Group smoking cessation programs are also an effective resource (214). Group formats have similar targets as individual counseling (modifying cognitions, motivation, and behavior). However, groups also offer opportunities for social learning and opportunities to practice new skills (233). Stead and colleagues (214) conducted a meta-analysis of 13 studies that compared group treatments to self-help materials. This analysis provides evidence that group programs are more effective than self-help materials (*RR* = 1.98, 95% confidence interval (CI = 1.60 - 2.46).

Smoking Quitlines

Similar outcomes have been observed in meta-analyses of smoking quitlines (128). Quitlines are telephone-based programs that help smokers with cessation. Quitlines provide a variety of resources to smokers including self-help resources (via the mail), counseling at the time of the call, and follow-up calls from the counselor (128; 144). There is evidence that participating in quitlines significantly improve smoking cessation outcomes when compared to minimal contact (i.e., self-help only or a single phone call;

(216). The pooled odds ratio for the eight studies in the meta-analysis was 1.40 (95% CI = 1.27 - 1.57).

In summary, research suggests that smokers have a variety of evidence-based treatments available. There are pharmacological treatments available that target the withdrawal and craving symptoms. Non-pharmacological treatments target cognitions, motivation, and behavior. Unfortunately, despite many smokers having the motivation to quit, cessation rates remain low. Recall that 6.2% of individuals maintain abstinence for at least six months (32). Therefore, there is a need for further research on smoking cessation treatments. As reviewed in more detail in the "Attentional retraining" section the current doctoral dissertation seeks to fill this research gap by testing an emerging treatment for smoking cessation.

Smoking Cessation among African American smokers

As described above it is very difficult for smokers to successfully abstain from smoking for long periods of time. African American smokers have lower rates of abstinence than Whites (222). Despite many African American smokers being motivated to quit, many quit attempts end in failure (32; 222). For example, the 1998 Surgeon General's report indicated that the prevalence of cessation is 35.4% for African American smokers compared to 50.5% for White smokers (223). More recent population surveys indicate that the odds of quitting are 44% - 49% lower among African American smokers compared to White smokers (70; 222).

African American smokers can select from the pharmacological and psychological treatments described above. There is evidence that nicotine replacement therapy, bupropion, and behavioral treatments improve cessation outcomes among

African American smokers when compared to control interventions (5; 233). However, African American smokers have poorer outcomes when compared to White smokers (221; 222). Table 1 lists the studies that have examined differences in smoking cessation among African Americans and Whites. The table includes studies that were published between 2000 and 2011. Ten studies were found that addressed this question (there were eight publications; Rabius and colleagues (182) and Piper and colleagues (175) each published two studies in one paper). Four of the studies involved the analysis of large-scale survey data, without a systematic tobacco cessation intervention. In these studies, cessation rates were compared for African American and White smokers. For instance, Trinidad and colleagues (222) conducted a logistic regression to examine the effect of race on cessation at six months utilizing data from the Tobacco Use Supplement to the Current Population Survey (n = 141,603). The odds of quitting for at least six months were 49% lower for African American smokers when compared to White smokers. Among the other three survey studies, two reported lower cessation rates for African Americans (70; 221). One study found significantly lower cessation rates among African Americans, but the difference was eliminated after controlling for sociodemographic differences (110).

Six studies assessed the effects of various interventions, including quit lines, nicotine replacement therapy, and bupropion. The authors of these studies also assessed whether race was associated with cessation outcomes. There were mixed findings among these studies. Four studies indicated that there were no differences in smoking cessation rates among African Americans and Whites (70; 174; 182). Conversely, two studies found smoking cessation differences between African Americans and Whites. Piper and

colleagues (174) examined racial differences in an efficacy trial of several pharmacotherapies including bupropion, nicotine lozenges, and nicotine patches (study 1). They found lower cessation rates among African American smokers (174). Similarly, Covey and colleagues (43) found lower cessation rates among African American smokers. In this study participants received an eight week trial of bupropion, the nicotine patch, and counseling.

It is difficult to draw conclusions about the effect of race on cessation in the intervention studies. Future research on racial differences in cessation rates for medication, quit lines, and counseling is necessary. Overall, this review suggests that differences in cessation among African Americans and Whites are detectable in large-scale survey studies. However, the mechanisms underlying racial differences in smoking cessation remain unclear.

Mechanisms of Poor Smoking Cessation Outcomes among African Americans

As described above, it is commonly reported that African Americans have lower rates of smoking cessation. Several possible mechanisms of the racial differences in smoking cessation have been studied, including the effect of menthol cigarettes, nicotine metabolism, tobacco withdrawal, and advertising. Advertising is the primary factor addressed in the intervention tested in this doctoral dissertation and is discussed in more detail later. First, a brief review of more commonly studied factors associated with poor cessation outcomes among African Americans are reviewed below.

Some evidence suggests that menthol use accounts for racial differences in smoking cessation (162). Research indicates that approximately 68% - 80% of African American smokers consume menthol cigarettes compared to 20 - 22% of White smokers

(77). The consumption of menthol cigarettes is believed to lead to greater exposure to nicotine because menthol allows smokers to inhale more deeply and smoke more of each cigarette (77). Specifically, several studies indicate that menthol cigarettes play a role in increasing the difficulty in quitting smoking among African American smokers (73; 162). For instance, studies indicate that menthol use moderates the relationship between race and smoking cessation (73). However, menthol use does not conclusively explain racial differences in smoking cessation outcomes (95).

Nicotine Metabolism Differences

Racial differences have also been reported in nicotine metabolism (28; 169). One study also reported that the half-life of cotinine, the major metabolite of nicotine which might be psychoactive (106), was 950 and 1,064 minutes for White and African American participants, respectively (169). Slower nicotine metabolism may allow for longer effects of nicotine (or cotinine) in the brain, thereby leading to greater dependence. There is evidence that differences in nicotine metabolism within and between ethnicities are associated with variations in the *CYP2A6* gene, the enzyme that converts nicotine to cotinine (156). There is also evidence that nicotine metabolism is inhibited by menthol (19) while other studies have found opposing results (194). Given that Africans Americans have a strong preference for menthol cigarettes, between-race differences in metabolism may be influenced by menthol. However, the research in this area is limited and inconsistent. The role of nicotine metabolism in smoking cessation is unclear.

Tobacco Withdrawal Differences

Tobacco withdrawal has also been considered as a factor that may explain differences in cessation (185). Although African American smokers consume fewer cigarettes per day than Whites, some laboratory studies suggest that African American smokers have higher levels of carbon monoxide and higher nicotine intake per cigarette (167; 169). Furthermore, African Americans prefer cigarettes higher in nicotine content (28; 34; 169). Given the higher nicotine intake per cigarette and preference for menthol cigarettes in African American smokers (noted earlier), it is possible that the brains of African American smokers are exposed to greater doses of nicotine.

Differences in tobacco withdrawal among African American and White smokers were explored in a sample of 203 smokers (185) .This study assessed subjective, cognitive, and physiological symptoms in a laboratory study with non-treatment seeking smokers. Tobacco withdrawal symptoms were assessed after participants smoked normally and following a 12 hour abstinence period. This study did not detect larger tobacco withdrawal symptoms among African Americans. This study suggests that it is unlikely that differences in withdrawal explain differences in smoking cessation.

Differences in Smoking Cessation: Additional Mechanisms

There are additional mechanisms that may explain differences in smoking cessation rates among African Americans and White smokers. There is evidence that African American smokers encounter several barriers to smoking cessation. They have unequal access to medical care (e.g., they are disproportionally uninsured (39) and are less likely to utilize Nicotine Replacement Therapy (72). However, as noted earlier, there is some evidence that African American smokers have poorer outcomes than White

smokers when receiving the same treatment (Nicotine Replacement Therapy, Bupropion, or Combination Therapy (43; 174). Conversely, African American smokers have some characteristics that should promote cessation. For example, they report that they make more quit attempts than White smokers and are more motivated to quit (72).

In summary, it is clear that the lower rate of smoking cessation among African Americans is not well understood. The studies thus far have investigated biological and behavioral differences among African American and White smokers. It is also possible that environmental factors such as smoking cues in the form of tobacco advertisements, may contribute to the difficulty experienced by African Americans. A critical review of smoking cues is presented to further elucidate the disparities in smoking cessation that exist among African American smokers. Furthermore, smoking cues are the target of the intervention tested in this doctoral dissertation study.

African American Smokers and Environmental Smoking Cues

Many African American smokers live in environments with a disproportionately high density of smoking cues, such as tobacco advertisements (13; 100; 190). This exposure may serve as an additional barrier to smoking cessation for African American smokers.

Point of Sale Advertisements

In African American communities, tobacco advertisements are typically displayed in retail storefronts or inside of the store. These types of advertisements are referred to as Point of Sale (POS) advertisements. The tobacco industry currently spends approximately $160 million per year on POS advertisements (60). This amount of money allows for aggressive marketing to African American smokers and other ethnic minority

smokers. The strategies tobacco companies have used to market to low-income and ethnic minority smokers have been well-documented (75). For example, reviews of tobacco industry documents and interviews (from the Legacy Tobacco Documents Library; http://legacy.library.ucsf.edu) have revealed a long history of tobacco companies targeting menthol brands to African American smokers. Tobacco companies began targeting African American smokers with culturally-tailored menthol advertisements in the late 1960's (9). As a result, menthol advertisements are overrepresented in African American neighborhoods and magazines (75; 123). Furthermore, the results of a meta-analysis investigating differences in advertising between African American and White communities revealed that the odds that any given advertisement was smoking-related was 70% higher in African American areas vs. White areas (181).

Effect of POS on Smoking

There is also an emerging area of research investigating the effects of POS on cessation and cigarette consumption. For example, data collected from smokers leaving retail stores in suggested that POS advertising is associated with unplanned purchases of cigarettes (35). In this study, the authors observed that 11.3% of purchases were unplanned. This evidence is consistent with other studies investigating impulse/unplanned purchases. For example, Wakefield and colleagues (226) found that 25% of a sample of 2996 adults reported purchasing cigarettes on impulse as a result of seeing cigarette displays. In addition, qualitative data suggest exposure to POS advertisements is associated with higher craving, smoking, and poorer rates of smoking cessation (90). This finding is consistent with a study that had smokers record their daily exposure to POS advertisements (26). This study revealed a positive association between

exposure to retail tobacco advertisements and smoking. A study by Reitzel and colleagues (183) also highlights the effect of advertising in retail outlets. In this study, a sample of ethnically diverse smokers was assessed to examine the association between proximity of tobacco retail outlets and cessation outcomes. Residential proximity to tobacco outlets was associated with cessation outcomes. Participants residing closer to tobacco outlets were more likely to relapse. Although exposure to POS advertisements was not directly investigated in this study, it is likely (in light of the above evidence) that individuals in close proximity to tobacco outlets had greater exposure to POS advertisements.

In summary, it is apparent that POS advertisements serve as powerful environmental cues to smokers. Notably, African American smokers have higher rates of exposure to POS advertising. Therefore, it is likely that they are at greater risk of relapse/smoking. In the section below a more detailed review of addiction theories is presented to elucidate the implications of the influence of smoking cues on drug use (see "attentional bias and addiction").

Related to the discussion above, the current study presents a model to summarize the relationship between cues and relapse/use. Figure 1 depicts the relationship between a cue rich environment and relapse/use ("route 1"). Additionally, addiction theory and research suggests that attentional bias to drug cues (briefly described earlier) is associated with relapse/use ("route 2"). Both attentional bias and a cue rich environment increase a smoker's exposure to smoking cues, thereby promoting relapse/use. A detailed review of the evidence for route 2 is presented below because this doctoral dissertation aims to reduce attentional bias in African American smokers.

Attentional Bias and Addiction

Many addiction researchers argue that responses to drug-related cues maintain drug use and undermine cessation attempts (e.g., (158; 189)). Particularly pertinent to the current application is the incentive-sensitization theory (ICT; (188)). As briefly noted earlier, this theory suggests that persistent drug use causes changes in the brain circuits that regulate the attribution of incentive salience to stimuli (189). This process causes pathological levels of salience to be assigned to drugs and drug cues. Stated another way, drug use causes drug-related stimuli (e.g., cigarettes) to become so desirable and attractive that they command attention and provoke approach behaviors. The phenomenon in which drug cues command attention is referred to as attentional bias. Berridge (23) described this phenomenon succinctly in the following statement:

"When attributed to a stimulus representation, incentive salience transforms the mere sensory shape, smell or sound into an attractive and attention-riveting incentive. Once attributed, the incentive percept becomes difficult to avoid noticing, the eyes naturally move toward the incentive, it captures the gaze and becomes motivationally attractive, and the rest of the body may well follow to obtain it. (p. 2)"

This statement evokes an image of the attention-grabbing properties of tobacco cues. Moreover, it highlights the process of how an object becomes motivationally attractive.

Attentional Bias and Psychological Theory

Attentional bias is part of a broader literature of cognitive psychological theories that have been applied to addiction. For instance, cognitive models propose that there are two distinct types of cognitive processes (207). There are fast, parallel, automatic

processes that are often outside of one's conscious awareness. Conversely, there are non-automatic, slow, reflexive, serial processes which are sometimes referred to as controlled processes (104). Attentional bias is an automatic process. For example, a smoker may attend to a smoking-related cue without making the conscious decision to do so (63). Understanding automatic cognitive processes such as attentional bias has become a growing area of research (63). For example, the approach of targeting attentional bias is consistent with a broader base of health behavior research that targets automatic processes (140).

Measuring Attentional bias

Attentional bias is a measureable phenomenon that can be assessed by reaction time tasks such as the visual probe (VP) task, described below, and the smoking Stroop task. In the latter, drug users (and controls) are asked to respond to neutral words and smoking-related words. Slowed responding on the drug-related words (versus the neutral words) is indicative of attentional bias. Attentional bias is associated with drug use. Specifically, drug users exhibit an attentional bias to drug cues and non-drug users do not (45). While reaction time measures have traditionally been used to measure attentional bias, bias can also be measured with eye tracking and self-report measures (127). The following review summarizes the reaction time measures, self-report, and eye tracking measures used in the current study.

Visual Probe Task

The visual probe (VP) task has been widely used to assess attentional bias in addiction and is used in current study. This task is grounded in research that indicates that individuals are faster to respond to a stimulus (e.g., a small dot) which is presented in an

attended (vs. unattended) part of a visual display (178). In this task a picture (or word) pair is presented on a computer screen. One member of the pair is located on the left side of the screen and the other is located on the right side. One picture is motivationally salient (e.g., a cigarette) and the other is motivationally neutral. The picture pair is presented for a brief duration (typically 500 ms), and when it disappears a probe (e.g., a dot) is presented in a position that had been occupied by one of the two pictures (or words). Participants are required to indicate the dot's position [left or right] as quickly and as accurately as possible.

A common finding in research using the VP task is that individuals are faster to respond to probes that replace motivationally salient stimuli (149). Researchers have inferred that attention has shifted toward the motivationally salient stimulus, suggesting that there is an "attentional bias" to the salient stimulus. That is, individuals can perform the classification task faster because their attention has already shifted to the position that the dot is presented. Numerous studies have used the VP task to demonstrate attentional biases in a range of disorders including anxiety (e.g., (135)), and drug addiction (e.g., (133)) including tobacco addiction (e.g., (229).

Self-Reports of Attentional Bias

Although the cognitive processes which give rise to attentional bias to drug cues operate largely automatically, there may be a conscious end product. For example, a smoker trying to abstain may find him or herself staring at a burning cigarette, although he or she would have no understanding of the automatic cognitive processes that caused the shift of attention in the first place. Likewise, on the VP task, the cognitive processes that drive the shift in attention to the salient stimulus occur too quickly for the

involvement of conscious processing, but the participant may (consciously) appreciate that he or she is attending to salient stimuli in the task. Although self-reported attentional bias is likely to be a crude outcome measure of the underlying automatic processes, self-report measures of attentional bias have shown promise (see preliminary studies). For example, the 8-item Subjective Attentional Bias Questionnaire (SABQ), which will be used in the proposed study, has shown favorable psychometrics properties (127). For example, the SABQ total score (mean of 8 items) was greatly increased by abstinence (effect size $d = 1.23$, $p < .001$), and the abstinence-induced increase in the SABQ was reliably correlated with a measure of tobacco dependence ($r = 0.24$, $p < .001$).

Eye movements and Smoking Cues

Another useful measure for assessing attentional bias to smoking cues is measuring and quantifying eye movements (151). Assessing eye movements allows for an objective assessment of attention to smoking cues. Studies of attention and addiction have included measures of eye movement to assess attentional processes in drug users. For example, Rosse and colleagues (192) assessed the visual scanning paths of cocaine dependent participants. Participants looked at an image of a crack cocaine pipe and a flower for 90 seconds each. The authors found that the cocaine dependent participants' visual scanning paths of the crack pipe more closely resembled the actual image than did the visual scanning paths of the flower. Additionally, accuracy of visual scanning distinguished heavy from light cocaine users. This study was one of the first to apply eye movement methodology to attention and addiction.

Eye movements have also been studied in attention to smoking cues. For example, Mogg and colleagues (151) examined eye movements in smokers who were administered

the visual probe task (VP). The authors assessed both the direction of eye gaze (initial orienting) and the duration of eye gaze (dwell time). The authors reported that smokers looked at smoking stimuli longer (versus neutral stimuli) when compared to non-smoking participants. Moreover, the amount of time that participants spent looking at smoking stimuli correlated with explicit measures of stimuli's affective and motivational valence ratings and with attentional bias. Differences in eye movements have also been observed in smokers during periods of withdrawal. Field and colleagues (67) found that smokers had a longer dwell time to smoking stimuli presented on the VP task during periods of withdrawal (10 hours of abstinence) when compared to non-smokers.

Attentional Bias and Relapse

Recent studies have revealed the clinical utility of attentional bias measures. For instance, a meta-analysis reported that higher attentional bias is associated with craving (65). Moreover, several studies indicate that elevated attentional bias to drug cues is prospectively associated with poor cessation outcomes in smokers, alcohol dependent individuals, methamphetamine dependent individuals, and cocaine users (29; 46; 138; 230). Table 2 provides a review of these studies. Three of the studies investigated attentional bias to smoking cues for smokers (99; 179; 230). These studies demonstrated that attentional bias can predict short-term abstinence; individuals with a higher attentional bias pre-quit or on quit-day had poorer cessation outcomes. Taken together, these studies highlight the clinical relevance of attentional bias and suggest that interventions that target attentional bias are warranted.

Current Study: Theoretical Model

Figure 2 depicts a more detailed model of the relationship between a cue rich environment and drug use/relapse ("pathway 1") and the relationship between attentional bias and drug use/relapse ("pathway 2"). (The term pathway is used for Figure 2 to differentiate them from the routes shown in Figure 1). In pathway 1 an individual's exposure to smoking cues in the real world is increased because of the volume of cues in a drug user's environment. This proposed mechanism is consistent with the literature on tobacco advertisements and retail outlets in African American communities. As depicted in pathway 2, attentional bias to smoking cues also increases exposure to real-world smoking cues. That is, attentional bias increases the number of smoking cues that smokers attend to in their natural environment, and the duration they attend to those cues. Addiction theory and research suggests that exposure to smoking cues (whether through a cue-rich environment or a high attentional bias) may directly cause relapse/use (pathway 3). Alternatively exposure to smoking cues may be mediated by craving (pathway 4 [exposure to craving] and pathway 5 [craving to relapse/use]; see Figure 2). The pertinent literature supporting these proposed mechanisms (pathways 3, 4, and 5) will be reviewed below.

The proposed model suggests that an intervention may occur by modifying: (1) the cue rich environment or (2) by modifying attentional bias. Because modifying the environment is difficult (modifying the environment would be costly and require the cooperation of tobacco companies), the author proposes to reduce exposure to smoking cues by reducing attentional bias (pathway 6). Attentional bias can be reduced through a reaction time task referred to as Attentional retraining. A review of the theory and

research on Attentional retraining is presented in the section "Attentional retraining" (see page 46).

Drug Cues and Drug Use

As noted earlier, exposure to drug cues may directly cause drug use/relapse (pathway 3). This "direct" pathway (or "habit" pathway) is supported by learning theories as well as empirical animal and human studies. An alternative mechanism is that exposure to drug cues causes craving (pathway 4) which, in turn, causes relapse (pathway 5). This mechanism will be referred to as the craving-mediated pathway. The pertinent literature on both the habit and craving-mediated pathway are reviewed below.

Drug Cues and Drug Use: Direct Pathway (Pathway 3)

As described above, drug cues are the target of the intervention tested in the current dissertation. Drug cues have been extensively studied in animal models and have been shown to provoke drug reinstatement, which is analogous to relapse (202). Moreover, studies have reported the ability of drug cues to reinstate nicotine seeking (27; 131). For example, in a study by Caggiula and colleagues (27), rats were trained to self-administer nicotine by pressing a lever over a twenty-day period. Nicotine infusions were coupled with a 1 second light cue and the turning off of a 1 minute house light cue (the house light was in the operant chamber). The training phase was followed by an extinction phase, which was similar to the training phase except responding (i.e., lever pressing) did not lead to nicotine. The authors observed that when nicotine cues were re-introduced, responding for nicotine returned to baseline levels within 5 days. The authors argued that visual cues are just as important as nicotine in the reinstatement of lever pressing after extinction. These findings have been replicated in several other studies (37;

131). These studies suggest that cues play an important role in relapse. This literature is consistent with the exposure literature in humans in which smokers report purchasing cigarettes in response to exposure to tobacco advertisements (90).

These studies are also consistent with other animal research which highlights the importance of drug cues. For instance, some researchers have argued that the Stimulus-Response (S-R) model of learning (also referred to as habit learning) dominates in the compulsion or habit aspect stage of addiction (55; 173). In the stimulus response model, behavior is under the direct control of a stimulus (or drug cue) that has previously been reinforced (91). For example, an animal will automatically press (the response) a lever (the stimulus) after it has been reinforced by receiving food pellets. A critical tenet of S-R learning that distinguishes it from other types of learning (e.g., goal-directed learning) is that the reinforcer is not encoded as a consequence or goal of the action. Instead the reinforcer serves to strengthen the stimulus-response association. Stated another way, the animal does not engage in the response (e.g., lever press) with the goal of receiving the pellets. Furthermore, this model suggests that the stimulus should elicit a response under circumstances in which the consequences are positive (receiving food) or aversive (e.g., a shock). For example, animal studies have demonstrated that rats will continue to press a lever to obtain food even if the reinforcer has been devalued (e.g., through taste aversion or satiety (3; 48; 147). Animal studies in which drug reinforcers were used instead of food have demonstrated similar findings (173). Researchers have demonstrated that habit learning occurs when animals have prolonged periods of training (e.g., 500 trials versus 100 trials (168). Prolonged training is believed to shift animals from goal directed behavior to S-R. This animal model is believed to be analogous to repetitive drug use. It

has been proposed that drugs that have short half-lives (e.g., nicotine) provoke the drug user to use more often and therefore develop more quickly into habits. For example, the short half-life of nicotine likely contributes to routine administration of nicotine among tobacco-dependent smokers (173).

In summary, this literature suggests that drug cues are important for relapse and also the maintenance of drug use. The animal literature is consistent with self-reports from humans about the influences of cues (or triggers) in maintaining smoking behavior and relapse. Taken together this literature highlights the need for interventions that target the influence of smoking cues on cigarette consumption and relapse.

Proximal and Distal Smoking Cues

There is a distinction between cues that are directly associated with a behavior (proximal) and cues that are present but not directly related to the behavior (distal) (Rudy (193). Animal studies in the spatial cognition literature have identified that separate brain regions are involved with processing proximal vs. distal cues which support this distinction (197). Proximal cues are defined as stimuli that co-occur with the goal stimulus and distal cues are cues that do not co-occur with a goal stimulus (193). The distinction between proximal and distal smoking cues has also been investigated in the smoking literature. Studies have examined proximal cues such as a lit cigarette and distal cues, stimuli that are regularly present with smoking but not directly to drug administration (41). Proximal cues and lighters are typically used to assess reactivity to smoking cues because they are commonly present across smokers. Studies have examined the effect of distal cues, specifically an environment linked with smoking, on craving (41; 42). Environments associated with smoking were associated with higher

levels of craving than non-smoking environments. Notably, these environments were void of proximal smoking cues, a factor that has confounded previous studies that investigated distal cues (31). Moreover, one study of distal cues has demonstrated that the magnitude of the association between distal cues and craving increases when the stimuli are personalized to the participant (41). The current study includes both proximal cues (e.g., picture of cigarette) and distal cues (picture of smokers).

Drug Cues and Drug Use: Craving-mediated pathway (Pathways 4 and 5)

An alternative mechanism that may explain the relationship between drug cues and drug use is craving. It has been argued that craving mediates the relationship between cue exposure and drug use (158) A discussion of craving in a broader sense is necessary to fully understand the empirical evidence for the craving-mediated pathway.

Craving

Craving can be defined as the conscious desire to use drugs (219). The concept of craving has been deemed clinically relevant as it is often seen as an expression of motivation for drug use (61). Craving has also been associated with drug relapse in many studies (62; 204).

Cue-induced craving

In the craving-mediated model, drug cues cause the drug user to experience craving which provokes drug use. This process is referred to as cue-reactivity or cue-induced craving. Many laboratory studies have studied the effects of proximal cues (e.g., a lit cigarette) on craving (31; 158). In these studies, participants are either exposed to imaginal cues or to images of cues. These studies have demonstrated that exposure to

drug cues reliably increases craving, when assessed by self-report and physiological measures (61). There is also evidence that cue-induced craving is clinically relevant. A brief review of the relationship between cue-induced craving and clinical outcomes is provided below.

Cue-induced Craving and Relapse

Cue-induced craving is associated with nicotine dependence (61). For example, there is evidence from prospective studies with treatment seeking smokers that reported that cue-induced craving can predict relapse (31; 191; 231). In these studies participants who were more sensitive to cue-induced craving were less likely to remain abstinent. There is also evidence from naturalistic smoking studies in which participants used electronic diaries. In these studies lapses were more likely to occur after exposure to smoking cues (206). Furthermore, there is evidence that cue-induced craving occurs weeks after quitting (206).

It should be noted however, that cue-induced craving will not always result in drug use/relapse. The conscious experience of desire to use allows drug users the opportunity to engage in other controlled cognitive process. Drug users can utilize coping strategies such as urge surfing or relaxation training to overcome the craving experience (61). The ability of drug users to engage in controlled cognitive processes may explain why there are mixed findings among studies investigating cue-induced craving and relapse (170). In addition, it is important to note that psychological cues are associated with smoking. For instance, changes in mood and stress are also associated with smoking (130; 165). However, the influence of emotional cues is beyond the scope of the current study.

In summary, this body of research highlights the importance of cues in addiction. Drug cues may elicit use/relapse either through a habit pathway or a craving-mediated pathway. It is likely that drug users are under the influence of both pathways during different time periods. Attentional bias is important for both pathways because it increases drug users' exposure to drug cues.

Assessment of Smoking Behavior

A comprehensive review of all measures of smoking is beyond the scope of this dissertation. In the current study, smoking behavior will be assessed using self-report, carbon monoxide levels in breath, and levels of cotinine in saliva. These measures are described in more detail later. However it is important to acknowledge that there are additional methods of assessing smoking.

Collecting salivary or plasma thiocyanate is an alternative measure of smoking behavior (16; 84). Thiocyanate is a metabolite of hydrogen cyanide which is delivered to the mouth from each cigarette. Smokers have levels of thiocyanate that are two to three times higher than non-smokers (38). Thiocyanate is also present in foods such as leafy greens and nuts and there is marked variation in thiocyanate levels among non-smokers (16). Although thiocynanate is highly correlated with smoking, it is not the best measure of smoking status particularly for individuals that are not heavy smokers or trying to quit (16; 84). The literature suggests that cotinine is better suited to study alterations in smoking behavior over time (84).

Smoking can also be assessed with tobacco specific nitrosamines such as 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone (NNK; (78)). NNK is metabolized in the body to NNAL 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanol (NNAL). NNAL has a

longer half-life (10-16 days) than cotinine (16 hours) and can be measured in blood, urine, and plasma. NNAL has been used to discriminate active versus passive smokers (people exposed to second hand smoke), to discriminate non-daily smokers vs. daily smokers (21) and to discriminate heaviness of smoking (103).

Pertinent to the current study, NNAL appears to have similar correlations (r = 0.478) with cigarettes smoked per day as cotinine (r = 0.426) (103). Cotinine was selected as a biomarker for the current study because the participant burden is low (no urine/blood samples were required).

Attentional retraining (Pathway 6)

As noted earlier both a cue rich environment and attentional bias contribute to drug use/relapse. While it is difficult to modify the external environment, it is possible to modify attentional bias (pathway 6). Attentional bias can be reduced by Attentional retraining (AR). AR refers to the use of modified tasks to change attentional bias (83), and is most often conducted using a modified visual probe (VP) task. The VP task has been modified to train participant's attention away from motivationally salient stimuli (e.g., drug-related stimuli). AR has shown promise in treatment of anxiety-related conditions (for reviews, see (83)).

In AR studies cognitive tasks (e.g., such as the VP) are modified so that they change (rather than assess) automatic processes. Typically, researchers train participants' attention toward or away from stimuli with the "modified" version of the cognitive task. Investigators then examine whether the intervention has had the desired effect on the automatic process by assessing performance on the "standard" version of the cognitive task. Investigators also assess whether the intervention influences other

outcome measures of interest, using other cognitive measures, self-report measures, and behavioral measures.

For example, researchers have used a modified VP task to train participants to automatically attend towards, or away from, negative stimuli. In the AR procedure used by MacLeod and colleagues (136), participants were trained toward (attend-negative) or away (attend-neutral) from negative stimuli. In the attend-negative group, the probe always replaced negative words. In the attend-neutral group, the probe always replaced neutral words. The training for both groups consisted of 576 trials. In this study, AR influenced attentional bias to negative stimuli, as assessed by the standard VP task (136). This was true for words that the participants did not receive training on and words that had been involved in the training. Moreover, attend-neutral participants reported significantly less anxiety and depression assessed after an anagram stress task compared with attend-negative participants.

AR has also been applied to addiction to reduce attentional bias to drug cues (65; 66; 198; 199). Field and Eastwood (66) demonstrated that the modified VP task could reduce attentional bias, reduce craving, and reduce alcohol consumption on a taste test. Schoenmakers and colleagues (199) reported that AR reduced attentional bias among alcoholic inpatients. Attwood and colleagues (11) demonstrated that AR could reduce attentional bias and craving in male smokers. However, no research has examined the efficacy of AR in specific racial/ethnic groups. Furthermore, many AR studies have either failed to report racial/ethnic characteristics or lack diversity (11; 198).

Therefore, the current study investigated the effect of AR in a sample of African American smokers. Fig. 3 illustrates the proposed effect of AR in African

American smokers. It was proposed that AR in African American smokers will reduce attentional bias, and therefore their exposure to smoking cues. After AR, their exposure to smoking cues should be reduced to a level more comparable with that of a smoker in a cue-lean environment. That is, although it is not possible to change the environment of an African American smoker, it may be possible to change their perception of the environment.

Attentional retraining on Mobile Devices

Mobile technologies can be leveraged to deliver AR in a naturalistic setting. Ecological Momentary Assessment (EMA) is a research methodology that can assess changes in attention and craving in the natural environment. EMA has been widely used to study smoking (203). In EMA, personal digital assistants or other mobile devices prompt participants to complete subjective assessments and cognitive tasks (137; 228). Recently, mobile devices have been used to deliver an AR intervention in a natural setting (52; 109). AR may be beneficial on mobile devices because more "doses" of AR may lead to greater reductions, or more sustained reductions, in attentional bias. Kerst and Waters (109) demonstrated that AR could be delivered for one week to smokers (described in more detail later). AR has also been administered on mobile devices in the anxiety literature (52). In this study, socially anxious participants completed 3 AR or control trainings daily on a smartphone for 4 weeks. Participants developed a bias away from threat-related stimuli which was larger in the AR group than in the control group. Symptoms of social anxiety decreased in both groups, but there no was no significant between-group difference. Taken together, these studies suggest that it is feasible to deliver AR on a mobile device.

Assessing Smoking Cue Exposure on Mobile Devices

In this doctoral dissertation study, environmental to exposure to smoking cues is assessed on the mobile device. Currently there is not a standard measure of environmental cue exposure (224). In some studies researchers have asked participants to retrospectively estimate their exposure to tobacco advertisements (200; 234). Exposure has also been assessed by having clients report their awareness of tobacco brands and by asking them to state their willingness to use tobacco promotional materials (224). Researchers have also assessed exposure by having the participant report their perception of the persuasiveness of the advertisement (200). These methods have been questioned because they are subject to recall bias and because they are not direct assessments of exposure. For instance, having a participant state their willingness to use promotional materials requires the client to evaluate the product and engage in cognitive appraisal (141). Despite these criticisms research suggests that there is a relationship between exposure and smoking particularly among youth (i.e. , youth initiation and progression to regular smoking (10; 49; 200).

There has been a recent interest in improving assessments of environmental tobacco cues (i.e., tobacco advertisements and tobacco in the media). Researchers have validated EMA methods to assess daily exposure to pro-tobacco marketing and media (141; 201). Martino and colleagues (141) investigated exposure to tobacco stimuli in a 21 day EMA study with college students (n = 134). This study assessed several types of tobacco cues including POS advertisements, billboards, tobacco use in movies, television, the internet, and direct mail. The participants were required to initiate an entry on the smartphone every time they encountered tobacco cues. The authors also utilized a retrospective survey of exposure (to assess the correlation between the two types of

assessments). There was not a significant correlation between the EMA exposure data and the retrospective assessment. The authors argue that this finding suggests that the measures capture different aspects of exposure. The authors also investigated the predictive validity of both the retrospective survey and the EMA assessment. They examined the relationship between the measures and smoking intention. The EMA assessment marginally predicted smoking intention and the retrospective assessment had no association with smoking intention. Furthermore, the authors reported that in a 3 week period there were 1,112 reported exposures among the college students which suggests that administering this assessment on a mobile device is feasible. In summary, this body of research suggests that it is important to assess exposure to smoking cues because exposure is associated with smoking behavior. Also, the studies indicate that it is feasible to assess exposure to tobacco marketing and media on a mobile device. The EMA methods of Shadel and colleagues (201) can be applied to studies of AR. To date, no AR studies have directly examined the effect AR on exposure to smoking cues.

Culturally Targeted Interventions

The Tobacco Use Dependence and Clinical Practice guidelines highlight the importance of the cultural appropriateness of smoking cessation treatments (68). The guidelines also emphasize the need for research on culturally targeted smoking cessation interventions for racial and ethnic minorities (68). Culturally targeted interventions are distinct from traditional treatments because they take into account characteristics shared by a subgroup. The rationale for using culturally targeted interventions is that utilizing subgroup characteristics increases the saliency of the intervention (117). There is evidence from the health promotion literature that culturally targeted interventions

maximize the effectiveness of treatments (116). Culturally targeted interventions are believed to improve effectiveness because they are viewed as more relevant to the subgroup and therefore more likely to be remembered. Culturally targeted treatments also allow researchers and practitioners to address barriers specific to the subgroup (e.g., menthol use among African American smokers; (184)).

To date, little research has examined culturally targeted treatments in smoking cessation (143). However, Matthews and colleagues (142) conducted a feasibility study of a culturally targeted group-based counseling program with nicotine replacement therapy. Their culturally adapted version (modified language, pictures, and statistics on print materials that were relevant to African American smokers) was compared to the standard treatment. Matthews and colleagues found that the culturally targeted treatment had higher retention, was perceived as more relevant, and had marginally higher nicotine adherence. This study suggests that African American smokers many benefit from interventions that take into account characteristics specific to them.

An AR intervention for African American smokers can be adapted to be more relevant to African American smokers. For instance, culturally-appropriate idiographic stimuli can be used for the AR and control procedures. Specifically, images of other African American smokers, menthol cigarettes, and tobacco advertisements could be used as the stimuli for the intervention and cognitive task. Furthermore, many AR interventions in the anxiety literature have customized the intervention to include words or pictures associated with specific type of anxiety (i.e., a variety of threat-related words for Generalized Anxiety Disorder and stimuli associated with specific phobias; (12)). Although, little research has assessed the effect of customized AR interventions, it is

logical that the AR intervention used in this doctoral dissertation study takes into account cultural differences (i.e., tobacco advertisements) and is representative of the targeted group.

Use of Mobile Devices among African Americans

As reviewed above, AR can be administered on a mobile device. In light of the research highlighting the importance of culturally appropriate interventions, it is noteworthy that administering AR on a mobile device has major implications for African American smokers. Mobile devices can help overcome barriers to smoking cessation treatments. For instance, African Americans are disproportionately low-income, which is a barrier to smoking cessation treatments (124). However, technological advances in mobile phones may provide a method of increasing treatment availability to African American smokers.

Mobile phones have penetrated the African American community at a higher rate than the general population (160). In 2011, 54.4% of African American adults owned mobile devices compared to 44% of Whites. Delivering AR trainings on mobile devices is an affordable way to deliver treatment to smokers who otherwise face financial barriers. Therefore, this technology may be particularly useful to African American smokers. As such the AR intervention in this doctoral dissertation study will be delivered to participants on a mobile device.

Preliminary Data

The author conducted a study assessing the relationship between attentional bias and smoking behavior among African American smokers prior to the doctoral dissertation. This study (study 1) was pertinent because there is a lack of research on

attentional bias among African Americans. The second preliminary study was the first study to deliver AR on a mobile device to smokers.

Study 1: Attentional Bias among African Americans (186)

Two preliminary studies of attentional bias in African American and White smokers were conducted (186). In both studies participants completed the Subjective Attentional Bias Questionnaire (SABQ; a self-report measure of attentional bias) at two laboratory visits, a non-abstinent session, and an abstinent session. In study 1, which involved non-treatment seeking smokers (99 Whites, 104 African Americans) the SABQ asked participants to report on experiences "so far today". Averaged across sessions, African Americans reported higher attentional bias on the SABQ ($p < .001$). In study 2, 110 Whites and 74 African Americans enrolled in a smoking cessation study and attempted to quit. Participants were followed from 2 weeks pre-quit through 4 weeks post-quit. Participants reported their experiences "during the past week" on the SABQ. As in study 1, averaged across sessions African Americans reported a greater attentional bias than Whites on the SABQ ($p < .005$). Higher attentional bias on the SABQ predicted relapse at Weeks 1 and 4 (CO Biochemical-verified Point Prevalence abstinence) ($p < .05$). In conclusion, African Americans reported greater subjective attentional bias than Whites, which may undermine cessation. At the very least, these studies suggest that attentional bias is an important component of dependence among African American smokers. The current study will build on this research by attempting to reduce attentional bias in African American smokers.

Study 2: Attention training in smokers (109)

In this study, sixty non-treatment seeking smokers (18.4% White; 61.7% African American; 20% "Other") were randomly assigned to an AR group or Control (no training) group. They carried a personal digital assistant (PDA) with them for one week. They were prompted to complete 3 attentional retrainings (AR group) or three control trainings (Control group) each day. The AR and Control groups completed 434 attentional retrainings (mean = 15.0) and 448 Control trainings (mean = 14.9), respectively. The two groups also completed 291 attentional bias assessments (using the VP task) in the field. Attentional bias assessed in the field decreased over time in the AR group, but not in the Control group. After day 5, there was a significant difference in attentional bias between the two groups, with the AR group exhibiting lower (more negative) attentional bias. No significant differences in attentional bias (on the VP task) were observed in a laboratory session at the end of training. Aggregated over all field assessments, craving after exposure to a picture containing both smoking and neutral information was lower in the AR group than the control group, suggesting that AR may reduce craving in response to a cue. There was no effect of AR on smoking behavior assessed using a smoking diary, and assessed with carbon monoxide and salivary cotinine. One limitation of the Kerst & Waters (109) study was that number of cigarettes smoked was not assessed on the PDA at each assessment. In the current study, an item was used to assess cigarettes smoked since the last assessment.

Specific Aims and Hypotheses:

Specific Aim 1: The primary aim of this study is to determine whether two weeks of AR delivered on a mobile device can reduce attentional bias in a sample of African American smokers.

Hypothesis 1.1: The AR group will exhibit a significantly lower (more negative) attentional bias on the VP task at the second and third laboratory assessments compared to the control group.

Hypothesis 1.2 The AR group will exhibit significantly lower attentional bias on the smoking Stroop task at the second and third laboratory assessments compared to the control group.

Hypothesis 1.3 The AR group will exhibit a progressively lower (more negative) attentional bias toward smoking-related stimuli over time, when assessed on the mobile device, compared to the control group.

Hypothesis 1.4: The AR group will report less attention capture by smoking cues (on the SABQ) assessed at the second and third laboratory session compared to the control group.

Hypothesis 1.5*:* The AR group will report less attention capture by smoking cues over time, when assessed on the mobile device, compared to the control group.

Hypothesis 1.6: During the mobile eye assessment, the AR group will look at the smoking stimulus for a smaller proportion of time than the control group.

Hypothesis 1.7**:** The AR group will report lower exposure to smoking-related stimuli over time, when assessed on the mobile device, compared to the control group.

Specific Aim 2: To examine the effect of AR on craving in African-American smokers.

Hypothesis 2.1: The AR group will report less craving on the Questionnaire for Smoking Urges at the second and third laboratory assessments compared to the control group.

Hypothesis 2.2. The AR group will report progressively lower cued craving over time, when assessed on the mobile device, compared to the control group.

Specific Aim 3: To explore the effect of AR on smoking in African American smokers

Hypothesis 3.1: The AR group will have lower levels of Carbon Monoxide at the second and third laboratory assessments compared to the control group.

Hypothesis 3.2: The AR group will have lower levels of cotinine in saliva at the second and third laboratory assessments compared to the control group.

Hypothesis 3.3 The AR group will have progressively lower rates of smoking over time, when assessed on the smoking diary and the PDA, compared to the control group.

CHAPTER 2: Methods

Overview

The current study examined the efficacy of AR administered on a mobile-device in reducing attentional bias, craving, and smoking. Non-treatment seeking African American cigarette smokers (N = 64) were randomly assigned to an AR or Control training condition. Participants were given a mobile-device for 2 weeks, which prompted them to complete up to three AR (or control) trainings per day. Participants completed assessments of attentional bias, craving, and smoking both in the lab and in the field (one time per day).

Participants

Sixty-four non-treatment seeking adult African American smokers were recruited from the Washington, D.C., area (see Figure 9 for Consolidated Standard of Reporting Trials Flow Diagram). Participants were recruited through newspaper advertisements and email lists (see Asppendix C). Participants were eligible for the study if they: (1) were aged 18 - 65; (2) self-reported as an African American; (3) reported smoking 5 to 10 cigarettes/day (light to moderate smokers; (6) or 11+ cigarettes per day (heavy smokers) for the past year; (4) had a home address and a functioning telephone number; (5) could speak, read, and write in English at an eighth-grade literacy level; and (6) specified English as the first language. Exclusion criteria were: (1) regular use of tobacco products other than cigarettes; (2) current use of bupropion, varenicline, or nicotine products, or currently trying to quit smoking; (3) another household member enrolled in the study; (4) self-reported color vision deficiency or unable to identify plates on the Ishihara test; (5) breath carbon monoxide (CO) <8 ppm for light and <10 ppm for heavy (6); or (6)

pregnant or breast feeding, (7) any other factor that, in the opinion of the investigators, would preclude completion of the protocol (e.g., not being able to adhere to study protocol). The inclusion and exclusion criteria were assessed during the phone screening and baseline laboratory assessment (detailed in "procedures"). Participants were compensated for participating in this study (see informed consent and IRB approval in the Appendix).

Procedures (Table 5)

Telephone Screening

Interested participants were screened on the telephone (see Table 5). Participants were provided a description of the study and were asked to provide verbal informed consent to be screened. The research assistant assessed the participant's eligibility for the study. Eligible smokers were scheduled for an in-person laboratory session (see Figure 9).

First Laboratory Visit

Eligible participants attended a baseline visit (Visit 1; see Table 5) where research staff provided a detailed description of the study, answered questions, confirmed eligibility, and obtained written informed consent (see Appendix B). Eligible participants performed several cognitive assessments, completed self-report measures, and received training on the personal digital assistant (PDA) as indicated in Table 5. Participants were instructed to respond when the PDA alerted them to complete a random assessment (RA). They were also informed that they could initiate assessments on their own (described in more detail later) but they would only be compensated for the RAs (PDA-initiated assessments).

To assess smoking behavior, participants provided a breath sample for carbon monoxide analysis (see section on "Measures"). They provided a saliva sample for analysis of cotinine levels (the major metabolite of nicotine; see section on "Measures"). Participants were told that they could "smoke as much or as little as they liked" during the two weeks. Individuals who declined to participate or were ineligible were provided with self-help materials and a referral to smoking cessation programs, if interested.

Participants also received training on the exposure assessment (described in the "exposure to smoking cues" section). Participants were given a smoking diary to take home with them. They were asked to record the number of cigarettes they smoked at the end of each day on the diary.

Randomization to Control or Training

During session 1, participants were randomized into the AR or Control group. Efforts were made to recruit similar numbers of light and heavy smokers. Participants were randomized to condition stratified by their smoking status (light vs. heavy). Light smokers were defined as individuals who reported smoking 5-10 cigarettes per day at telephone screening Heavy smokers were defined as individuals who reported smoking 11+ cigarettes per day at telephone screening Randomization.com was used generate the assignments. Both the participant and the research assistant were blinded to the condition assignment.

Week 1: PDA

All participants carried a Hewlett-Packard IPAQ PDA with them for the first week. Participants completed up to four PDA field assessments which included the training tasks (AR subjects), control tasks (Control subjects), and assessment tasks. At

three PDA field assessments they completed trainings (AR or control). At the fourth PDA field assessment, participants were prompted to complete the VP assessment (see section on attentional bias). Figure 6 provides a schematic depiction of when the participants completed the AR or control procedures and the assessment tasks.

As noted earlier, participants completed two types of PDA field assessments, RAs and participant-initiated assessments. For RAs, participants were prompted at four random times per day by the PDA to complete an assessment. Participants were also instructed that they could complete a participant-initiated assessment if they failed to complete an RA. Participants were informed that they would not be paid for participant-initiated assessments and that completing them was optional.

Second Laboratory Visit

After one week of training, participants returned to the laboratory. The cognitive and self-report assessments were re-administered as indicated in Table 5. Also, participants' smoking status was assessed (with saliva and breath samples). Research assistants ensured that all data had been retrieved from the PDA. Finally, participants completed the first eye tracking assessment (see section on Mobile eye tracking and Figure 5).

Week 2: PDA

Participants again carried a PDA for one week. As before, they were prompted at random times to complete three trainings per day (AR or control) and an assessment task. Again, participants had the option to complete participant-initiated assessments. The procedures for the second week were the same as those for the first week.

Third Laboratory Visit

At the final laboratory session, participants again completed the cognitive assessments and self-report measures. They returned the PDA to the research staff. Research assistants ensured that all data were retrieved from the PDA. Their smoking status was assessed (i.e., saliva and breath samples). Participants completed the second (final) eye tracking assessment (see section on Mobile eye tracking and Figure 5). After completing the study protocol, participants completed a post-treatment interview questionnaire.

Measures (Appendix A)

Nicotine Dependence

Nicotine Dependence was assessed by the Wisconsin Inventory of Smoking Dependence Motives (WISDM). The WISDM is a 68-question survey designed to assess smoking dependence based on 13 sets of motives (175). The total WISDM score ranges from 13 to 91 with higher scores indicating greater levels of dependence. Internal consistency for the total scale is excellent (Cronbach's α = .97 - .99) (175). Participants completed this measure at the baseline session.

Participants also completed the 6-item Fagerström Test for Nicotine Dependence at the baseline session (FTND) (87). The FTND has been validated (content and predictive validity (53) in a number of studies and has a Cronbach's alpha of .65 (177).

Attentional Bias

Attentional bias was assessed using the standard VP task on one PDA assessment of each day (Table 5). In a VP task (see example in Figure 4), a series of picture pairs (one motivationally salient and the other neutral) are presented relatively briefly (500 ms)

on a computer screen, with one picture on the left and the other on the right. When the pair disappears, a probe is presented in the position formerly occupied by one of the pictures. Participants were required to respond as quickly and accurately as possible to the probe. Typically, individuals are faster to respond to probes that replace motivationally salient stimuli (vs. neutral stimuli) because attention has shifted towards the salient stimuli. A stimulus duration of 500 ms was used because AR has been shown to be effective using this stimulus duration (199). Each assessment (lab or field) included 80 trials; on 40 trials the probe replaced the smoking picture, and on 40 trials it replaced the neutral picture. After the VP task, the participant was asked how many times he or she was interrupted while performing the task. The response options were: No times; one time; two times; three times; four or more times.

It is important to demonstrate that the effect of cognitive retraining generalizes to new stimuli and to new tasks (134). Therefore, in each laboratory assessment smoking stimuli were included on the VP task on which the participant had not received training (untrained or "new stimuli", described in more detail later). This allowed a determination of whether the effect of AR generalizes to new stimuli. In the field, one assessment per week involved stimuli on which participants had not received training.

The Smoking Stroop task is another reaction time measure of attentional bias (230). In this doctoral dissertation 33 trials of smoking words and 33 trials of neutral words were administered at each laboratory visit. This task allowed us to determine whether an effect of AR can be observed on a different attentional bias task. Participants were told that a series of words were presented on the screen, one after the other, and that the task was to indicate the color of the word as quickly and as accurately as

possible. They were told that they could ignore the meaning of the words; they were just required to respond to the colors. Processing of reaction time data and computation of attentional bias followed existing procedures (e.g., (227; 230)).

Attentional bias was also assessed with the Subjective Attentional Bias Questionnaire (SABQ). The SABQ is an eight-item questionnaire that assesses the extent to which participants notice that their attention is captured by cigarettes and smoking cues ((126), and has been shown to be useful in the preliminary data (described later). As noted in Leventhal (126), the SABQ has been shown to have good internal reliability (alpha = 0.88). In addition, the total score (mean of eight items) was increased by abstinence (effect size d = 1.23), and the abstinence-induced increase in the SABQ was significantly correlated with FTND scores (r = 0.24).

Self-reported attentional bias was assessed on the PDA at each assessment using a single-item: "Since the last assessment, my attention has often been drawn to cigarettes"; 1 = strongly disagree, 7 = strongly agree. Ratings on this item have been shown to be associated with attentional bias on the modified Stroop task (232).

Craving

The 10-item Brief Questionnaire of Smoking Urges (henceforth: "QSU" (44) was used to assess craving in the laboratory. This questionnaire assessed the intention and desire to smoke (Factor 1), and desire for smoking to reduce negative affect (Factor 2). A total craving score was computed and used in the current study. This measure has high reliability for both factor 1 and factor 2 (Cronbach's alpha = .89 and .87, respectively; (44)).

On the PDA, craving for cigarettes was assessed on a 7-point Likert scale (1= no craving, 7 = extreme craving) at each RA. Following Kerst and Waters (109), a second craving item was used. A picture with both smoking and non-smoking content, randomly selected from a pool of 58 pictures, was presented for 1 second. Participants subsequently reported their craving (1-7 scale). If AR causes attention to be drawn to neutral stimuli in the picture (as hypothesized), then "exposure" to the smoking content should be reduced and there should be reduced craving on this item.

Cigarette Smoking

Participants entered the number of cigarettes smoked each day on the smoking diary (Appendix A). Smoking was also assessed on the PDA using the following item: "Since the last assessment, how many cigarettes have you smoked", Response options were: None; 1 cigarette; 2 cigarettes; 3 cigarettes; 4 or more cigarettes.

Heaviness of smoking was also assessed with salivary cotinine (the major metabolite of nicotine; (166). Salivary cotinine can be considered the "gold standard" for measuring nicotine exposure (79). The participants were instructed not to eat or drink 10 minutes before sampling. They were offered a moist towelette to clean their hands and mouth (if necessary). Using gloves, the author or research assistant opened the vial and gave the participant the cotton roll. The participant was asked to place the cotton piece in his or her mouth and to gently roll the cotton piece in his or her mouth for a whole minute to saturate with saliva. The participant was requested to place the cotton piece on the edge of his or her mouth and re-insert it into the vial without touching the vial. Using gloves, the author tightly replaced the cap on the vial. Next, the author centrifuged the vial filled with saliva. The author removed the cotton piece container from the vial and

replaced the cap tightly over vial. The author then applied freezer tape over the label and labeled the vials legibly using a permanent marker (Sharpie-brand marker) and bar-code labels provided by Salimetrics. Samples were then shipped to Salimetrics to be analyzed. Salimetrics utilized the High-Sensitivity Cotinine EIA kit to analyze data (see appendix A).

Exhaled CO levels were assessed using a CO monitor (20). Participants were told (in the informed consent form) that they would be asked to complete a breath test and to provide a saliva sample at each laboratory visit. Exhaled CO levels were measured with a CO monitor (Bedfont Micro Smokerlyzer, Harrietsham, England), according to the manufacturer's instructions (see Appendix A for instructions). Exhaled CO levels are commonly used to validate recent abstinence from smoking monitors (20). The participant's CO level was obtained at the beginning of each experimental session. The CO monitor was calibrated from a cylinder of research gas with a known CO concentration (about 50 ppm) every 6 months as specified by the manufacturer.

At the baseline session (visit 1), if the CO monitor indicated that a participant's expired CO level was low (less than 8 parts per million (ppm) for light smokers, less than 10 ppm for heavy smokers) (6), he or she was excluded from the study. Participants were excluded because, if their expired CO level was below these levels, there is serious doubt as to whether the individual actually smokes at their stated rate (20).

Exposure to Smoking Cues

Exposure to smoking cues was assessed by a modified version of the methods of (201). Participants were shown slides of pro-tobacco cues including advertisements in magazines, advertising on the outside of storefronts, and POS advertising in stores.

Participants were also trained on how to complete the assessment on the PDA. Participants were asked to enter their responses in a forced-choice format (see self-reported exposure assessment in the appendix A). During the 14 day period, participants were asked to respond to these items at each PDA field assessment (see Appendix A, Measures). The items are on an ordinal scale (1= No advertisements seen, 2 = 1 advertisements seen, 3 = 2 advertisements seen, etc.). It was estimated that each exposure assessment in the current study would take approximately 1 minute. Evidence suggests that scores on the exposure assessment are marginally associated with future intention to smoke (201). The measure is also moderately correlated (r = .37) with a retrospective measure of tobacco exposure (201). The retrospective assessment was not used in the current study).

Other EMA items

At each PDA field assessment participants responded to the following items on 7-point Likert-type scales according to how they feel "right now"; Difficulty concentrating; Overall mood; Energy/arousal levels. In addition, three items assessed testing/lighting conditions; two items assessed context (e.g., location); two items assessed recent alcohol/coffee intake; and two items assessed the recency of the last cigarette smoked (227).

Two items assessed purchases of cigarettes: "Since the last assessment, have you purchased any cigarettes "on impulse"? (Yes/No); and "Since the last assessment, have you purchased any cigarettes at all? (Yes/No).

Intervention

AR participants were scheduled to complete three AR tasks per day. On the AR tasks, the dot always replaces the neutral picture. Therefore there is a perfect association between picture type and dot location. Control participants were scheduled to complete three control tasks per day. On the control task, the dot is equally likely to replace the smoking picture and the neutral picture. Therefore there is no association between picture type and dot location. This type of control condition has been used in previous AR studies (e.g., (65; 109)). It ensures that the duration of AR and control training should be similar. In addition, AR and control participants receive equal practice on the VP tasks and are exposed to the same pictures (smoking and neutral). Based on previous studies, the mean duration of AR and Control training assessments was expected to be about 7 minutes.

Mobile Device Hardware and Software

The Hewlett-Packard IPAQ Personal Digital Assistant (PDA) runs on the Windows Mobile Operating system. Application programming was done in C#.NET by Terminal C, a Houston-based company. Participants used the PDA keys ("hard keys") and touch screen to enter their responses. Participants completed the self-report items in a manner that is similar to a paper-and-pencil questionnaire. They used their fingers or a stylus to select from a list of responses. As with other EMA studies, the participants were locked out of all other functions of the PDA.

At each PDA field assessment, the first assessment was the cue-provoked craving item. An image with smoking and neutral stimuli was displayed on the screen for 1 second, and then the cued craving item was presented. After the cue-provoked craving measure, the other self-report questions were administered, including assessments of

mood, exposure to smoking cues, attention to smoking cues, smoking behavior, and purchasing behavior. During these questions the second non-cue-provoked craving item was presented. Following the self-report items, instructions for completing the VP task were presented. The instructions were immediately followed by the VP task (AR task, Control task, or standard VP assessment). At the conclusion of the VP task, the participant was asked how many times he or she was interrupted during the completion of the VP task, as noted earlier. Figure 6 presents a schematic depiction of a PDA field assessment.

Stimuli

Pictures for VP Task

The pictures for the training/control task and the VP task were selected from pictures stored on Flicker and pictures created by the author (Fig. 7). Permission was obtained from the photographers and a photography credits sheet was provided to participants. Because there was a lack of pictures of African American smokers on Flicker, many of the photographs were created by the author. Permission was obtained to take pictures of research staff members and USUHS graduate students. The author and research staff also created many of the cue provoked craving pictures to ensure the pictures depicted one smoking stimulus and one neutral stimulus.

There were 1,141 pictures selected from Flicker or created by the author. The best pictures were selected from this pool by evaluating valence, noticeability of smoking stimulus (if present), and a global judgment rating which evaluated the suitability of the pictures for the current study. Two independent raters who were knowledgeable of the study design rated each picture using a scale of 1 to 7. The items were: “How

negative/positive is this picture to you? (1=extremely negative; 4 = neither positive nor negative; 7 = extremely positive); "How noticeable is the smoking content in this picture" (1=Not noticeable at all, 7=Highly noticeable); "Overall, based on your knowledge of the study, how suitable is this picture for the study" (1=The picture does not work for the study, 7=The picture should work very well for the study). A pictures was used for this study if it had a "7" (maximum score) on the noticeability and suitability questions, and a non-extreme score on the valence question. From this pool 80 smoking object (SO), 80 neutral ("non-smoking") object (NSO), 80 smoking human (SH), and 80 neutral ("non-smoking") human (NSH) pictures were selected. The 320 pictures were randomly assigned to be included in one of 16 picture lists. There were 14 lists used to ensure that there was a unique list for each day in the study and 2 lists of "new" pictures (described later). Each picture list contained 20 pictures, 5 smoking objects, 5 neutral objects, 5 smoking human, and 5 neutral human. The smoking object and neutral object pictures were randomly paired together. Likewise, the smoking human and neutral human pictures were randomly paired together. The smoking picture was equally likely to be on the left or on the right. The 20 picture sets were utilized to create a file of 160 trials (20 picture sets repeated 8 times) for control/training and 80 trials (20 picture sets repeated 4 times) for the assessments presented on the PDA. For the field training a spreadsheet was created for each participant which contained 160 trials repeated 3 times for the trainings/control and 1 set of 80 trials for the assessment. This process was repeated for each day of the study so that the participant's file contained 42 trainings/control with 160 trials and 14 assessments with 80 trials.

New (or "untrained") pictures for the PDA were created from a set of pictures unique from the 14 field lists described above. The list consisted of pictures that participants were not scheduled to receive training on (AR or Control training) in the field. The new pictures were organized to create 80 assessment trials following the assessment procedures described above. Each participant saw a new picture list once during week one and once during week two. Thus on the day during which the participants saw the new picture list they were presented with three AR or control trainings as usual and a 80-trial assessment with pictures from the new list. Thus the assessment occurred using pictures on which they had not been trained.

For the three lab assessments each participant was presented with 160 trials. During visit 1, all participants were assessed on one particular list (picture list 11) which was selected at random from the lists. During visit 2, participants were assessed with a picture list on which they were scheduled to receive training ("old" pictures). Participants were also assessed with the untrained ("new") pictures used in the first week of the field assessments. Similarly, during visit 3 participants were assessed with a picture list on which they had been scheduled to receive training ("old" pictures), and they were also assessed with the untrained ("new") pictures used in the second week of the field assessments. Thus, at the lab assessments, participants were assessed on pictures on which they were trained (referred to as "old pictures") as well as pictures on which they had not been trained (referred to "new pictures") (but that they may have previously seen during an assessment).

Pictures for Cued Craving

Research staff also rated cued craving pictures using the three questions described above. The cued craving pictures consisted of humans or objects with smoking and neutral features (see Appendix D). The cued craving pictures were selected from Flicker or created by research staff. There were a total of 63 cued craving pictures generated, and 58 pictures were selected using the same procedures as described above. A cued craving picture was randomly selected to appear during each PDA field assessment.

Pictures for Mobile Eye

The mobile eye pictures (Appendix D) consisted of one smoking and one neutral advertisement, side by side. The mobile eye pictures were selected from magazine advertisements. There were a total of 36 mobile eye pictures developed. Three pictures were selected at random from the pool of 36. A picture was randomly assigned to be presented at each mobile eye assessment, under the constraint that each subject saw each picture only once.

Mobile Eye tracker

As noted in the introduction attentional bias to smoking cues can also be assessed with eye tracking (108). The Applied Science Laboratories (ASL) Mobile Eye system was used to assess participant's attention to smoking cues in the laboratory at visits 2 and 3. This system has been used in a number of research studies and a previous study of AR (e.g., (108; 180)). The ASL eye system was selected for the current study because it can collect eye movement data while a participant is mobile. The ASL mobile eye system is compact with eye tracking optics that are lightweight and relatively unobtrusive. This design allows for data collection during natural movements. The system includes a

spectacle mounted mini camera and monocle that reflects a corneal image illuminated by infrared three LEDs to capture eye gaze. The system also includes a second spectacle mounted mini camera that captures the scene gaze. The eye and scene images are recorded to a small DVCR system. The DVCR is battery powered and carried by the participant in a small hip pack. The eye image and scene image are automatically interleaved and saved on a DVCR tape. The video is then transferred to a password protected laptop where it is saved as a video for later analysis using GazeTracker software. Gazetracker is designed by Eyetellect, a company that specializes in software for analyzing mobile eye tracking data. The image (scene and eye image) is separated and a scene video can be created with a variable cursor overlay. The gaze location is recorded at close to 30 Hz and mapped to the 640 X 480 pixel display scene video. This combination of data allows for comparison between the scene video and the relative gaze location.

The methods for the eye tracking were developed from technical consultation with ASL and adapted from Kerst (108). During the second laboratory session, the participant was fitted with adjustable glasses in the main laboratory room (Building 28-101). A research assistant calibrated the system for each participant. The calibration process included having the participant look at 9 photographs that were on the wall while wearing the mobile eyeglasses. Participants were instructed to look at each photograph for 3 seconds to ensure that the glasses were appropriately tracking their eye movements. As recommended by ASL, participants were calibrated at a distance of approximately 4 feet from the calibration points.

After calibration, the researcher led the participant into another research room (28-102). The researcher informed the participant that he or she (the researcher) needed to set up a task in the cognition lab (28-101). In 28-102 there was a smoking stimulus (unlit cigarette, cigarette pack, and lighter) on the desk. Other items in the room were neutral and included: a computer; an empty coffee mug; a small plant; an air purifier on the ground; a desk chair; a telephone; and artwork on the wall (see Figure 5). The participant was instructed to have a seat in the chair. The researcher left the participant in the room for 1 minute. During that time, the participant was free to look around the room. Participants were then escorted back to the cognition lab (28-101). To conceal the true purpose of the eye tracker assessment, the researcher then asked the participants to look at a poster-sized image of two advertisements. The advertisements were side by side and included 1 smoking advertisement and 1 non-smoking advertisement. Analysis of the eye movement data for these advertisements was beyond the scope of the current study. The main purpose of this assessment was to disguise the eye tracking assessment that occurred in the smoking laboratory (in room 28-102). Eye tracking data were collected at visits two and three only. Baseline eye tracking data were not collected to ensure the novelty of the task, at least at visit 2. It is plausible that bias to smoking cues could be attenuated if participants were repeatedly exposed to the staged environment in the smoking room. Similarly, if the participant completed the assessment at baseline, knowledge of the task (including the presence of the cigarette) could potentially influence attentional processes in subsequent tasks in a way that could potentially undermine the utility of the task.

The methods for the mobile eye assessment at visit three were the same as visit two except participants were debriefed about the purpose of the mobile eye assessment. Specifically, the researcher informed them: "As you know from the consent form, the purpose of this behavioral research study is to evaluate a new method of influencing smokers' attention, cravings and smoking. When you wore the spectacles, we wanted to know at what you were looking. If one of the training conditions changes how your attention works, it may influence how long you spent looking at different objects in the smoking room or the order in which you looked at them. By measuring your eye movements, we can test if the training conditions influenced this aspect of your attention."

To analyze the mobile eye data, two independent raters (the author, and a post-doc in the laboratory) coded the data from the videos in the Gazetracker software. The primary purpose of the coding process was to create a smoking lookzone so that the amount of time spent looking at the smoking stimulus could be calculated. A lookzone, or area of interest, is defined as a manually drawn user defined area placed over stimuli in a particular area of interest on the scene video. For this study raters were instructed to create a lookzone which completely surrounded the smoking stimulus. The defined lookzones were manually resized and moved around the recorded scene video as participants moved about the room (see Figure 5 for an example of gaze location and lookzone captured during coding with example outcome data). When smoking stimuli were not on screen the lookzone was moved to a neutral area outside of the recorded scene video so that no gazepoints could be recorded in the lookzone when smoking stimuli were not visible. To accomplish this task the raters were instructed to slow the

videos to 1/10th of the normal speed and stop as necessary to make adjustments to the size of the lookzone.

As noted in the CONSORT diagram, there were missing data due to malfunction of the mobile eye device. The analysis included data from 34 participants at session 2 (20 AR participants and 14 Controls) and 32 participants at session 3 (19 AR participants and 13 Controls). Therefore a total 66 videos were available for analysis.

Gazetracker provided data on the following outcome variable: gazepoints in look zone. (Each gazepoint was 33 ms in duration). To ensure the data were reliable, videos in which discrepancies (defined as a between-rater difference of 10+ gazepoints) were identified and underwent additional review. For these 17 videos, two independent raters (the same raters) viewed the videos again. In the second viewing the raters compared the output from gazetracker to the movement of the fixation cross in the video. The rater then selected the gazetracker output generated from the previous ratings that best reflected what was actually observed in the video. A Cohen's Kappa for this decision (rater data to use) was 0.87 meaning the two raters agreed in how to resolve the discrepancy. This additional validation ensured that the gazetracker output reflected what the participant was actually looking at. For the other 49 videos, a discrepancy between the two raters was not present. For these 49 videos, the correlation between raters for the number of gazepoints in the smoking lookzone was r = .82. Because errors in gazetracker processing tended to reduce the number of gazepoints identified by the software, the number of gazepoints used in analysis was the higher number of the two raters. Results did not vary in the mean rating were used.

The primary outcome variable was the number of gazepoints in the smoking lookzone. If AR causes participants to attend away from smoking stimuli, as hypothesized, then it was expected that participants in the AR condition would gaze at the cigarette for a briefer duration (i.e., have fewer gazepoints in the smoking lookzone) than those in the control condition.

Color Deficiency

Color deficiency was assessed because the smoking Stroop task requires participants to respond to colors which include red and green. Red-green color deficiency was assessed during the telephone screening by self-report and objectively at the baseline session. The Ishihara test is the most widely used measure for color deficiency (Birch, 1997). During the baseline session participants were required to look at Ishihara plates. Participants who were unable to identify the numbers embedded in the plates were excluded from the study. One participant was excluded based on performance on the Ishihara test.

Post Treatment Questions

Blinding Manipulation Check

The effectiveness of blinding to treatment condition was assessed at the final visit using the following question: “There were two treatment conditions in this study. In the active condition, the experimenter was trying to manipulate your attention so that your craving for cigarettes would be reduced. In the control (or inactive) condition, there was no attempt to manipulate your attention. You were assigned to one of these two treatment conditions. Which condition do you think you were in?” (Response Options: “Active/” or “Inactive.”)

Acceptability of Treatment

Acceptability of treatment was assessed at the final visit. Participants completed a questionnaire including the following items: "Did you find this intervention acceptable?" and "How likely are you to recommend this treatment to a friend?" The participants were also asked to rate how boring they found the intervention (1 to 7; 1 Not Boring at all to 5 Very Boring).

Compensation (Appendix B)

Non-federal civilians received $20 for completing the orientation session (even if ineligible), and $20 for completing the second laboratory and third laboratory session. Non-federal civilians received $1 for each mobile device assessment that they completed. They also received $3 for each day (except the final day) that they contributed data to the study, up to a maximum of 14 days. We also advertised to federal civilians and military personnel. Per the Institutional Review Board guidelines, federal civilians and military personnel would receive compensation for the laboratory sessions and the mobile device assessments that occurred during non-duty hours. However, no military or federal civilians were enrolled in this study.

Analytic Plan

The criteria for excluding data follow the procedures of Kerst and Waters (2013). For EMA data, individual (trial-level) RTs from incorrect responses (0.43% of trials) were excluded as were individual (trial-level) RTs less than 100 ms on correct responses (0.002% of RTs on correct responses). Median RTs (on correct responses) were used to reduce the influence of outliers. As noted earlier, at each assessment attentional bias was computed as the difference in median reaction times to respond to probes (i.e., indicate

the location of the probe by pressing the left or right button) that replaced smoking and neutral pictures. For example, if on assessment 1 participant 1 had a median RT of 550 ms to respond to probes that replaced smoking pictures and a median RT of 600 ms to respond to probes that replaced neutral pictures, the attentional bias score for assessment 1 (for participant 1) would be 600 ms - 550 ms = +50 ms. A positive attentional bias scores therefore means faster responses to probes that replaced smoking pictures than probes that replaced neutral pictures, meaning that attention has shifted toward the smoking picture. If on assessment 2, for example, participant 1 had a median RT of 600 ms to respond to probes that replaced smoking pictures and a median RT of 550 ms to respond to probes that replaced neutral pictures, the attentional bias score for assessment 2 would be 550 ms - 600 ms = -50 ms. A negative attentional bias score therefore indicates faster responses to probes that replaced neutral pictures than probes that replaced smoking pictures, meaning that attention has shifted away from the smoking picture. This is termed "avoidance".

Bias scores from assessments on which participants made more than 25% errors (2 assessments, 0.37% of assessments) were excluded from analysis, as were bias scores more extreme than +1000 ms or -1000 ms (> 5 SDs from mean) (2 assessments, 0.37% of assessments). On the "interruption" question, participants reported the following responses (AR vs. Control): No times = 78.1% vs. 78.2%; 1 time = 11.5% vs. 12.9%; 2 times = 4.2% vs. 5.5%; 3 times = 2.7% vs. 2.6%; 4+ times = 3.5% vs. 0.7%. There was no effect of Group on reported interruptions. Consistent with Kerst (2013), mean RTs (average RT on neutral and smoking trials) were slower on trials with more interruptions, $F(1, 425) = 13.67$, $p = .0002$. For example the mean RT for "no interruption"

assessments was 766 ms whereas for "4+ interruption" assessments it as 980 ms. Therefore, the primary analyses used data from assessments in which participants reported no more than 1 interruption (481 assessments, 90.4% of assessments with bias data). EMA data from the first training completed in the field were also excluded from analyses because these data were obtained prior to intervention.

For the lab assessments, assessments on which participants made more than 25% errors (2 assessments, 1.29% of assessments) were excluded from analysis, as were bias scores more extreme than +300 ms or -300 ms (> 5 SDs from overall mean) (2 assessments on "old" pictures, 1.29% of assessments). (On "new" pictures, no bias scores were more extreme than +300 ms or -300 ms). Consistent with the EMA data, assessments in which participants reported two or more interruptions were excluded from analysis (8 assessments, 5.0% of assessments). Missing data at session 1 were replaced by the group mean when the session 1 data were used as a covariate for the analysis of EMA data.

On the smoking Stroop task, data were excluded on assessments in which participants made more than 25% errors (17+ errors) (1 assessment, 0.64% of smoking Stroop assessments). Smoking Stroop scores more extreme than +700 ms or -700 ms (> 5 SDs from mean) were also excluded (1 assessment, 0.64% of smoking Stroop assessments).

Overall Analytic Plan

Linear mixed models (LMM) (PROC MIXED in SAS; (129)) were used for the primary analyses. These analyses allow for the fact that subjects differ in the number of

observations available for analysis, and take into account the clustering of data within subjects. All tests were 2-tailed, and alpha was set to .05.

To analyze EMA data, Day in study (a within-subject continuous variable) was entered as a continuous variable, along with Group (a between-subjects variable with 2 levels, AR vs. control). The Group by Day interaction term was also tested. A random (subject-specific) intercept and an autoregressive model of order 1 for the residuals within subjects was used. The within-subject variable, Day, was treated as a random effect in the model if the *p*-value for the covariance parameter estimate (for Day) was less than 0.1 (69). Time of day (continuous variable) and Assessment Type (RAs vs. participant-initiated) were also added as covariates. Each dependent variable was tested in a separate model. Baseline measures of each dependent variable were included as a covariate.

To examine the effect of Group on data collected in the laboratory, LMMs were also used, with one between-subjects factor (Group: 2 levels) and one within-subject factor (Visit) with two levels post-intervention (Visit 2 vs. Visit 3) included in all models. The Group by Visit interaction term was also tested. As with the analysis of EMA data, each dependent variable was tested in a separate model, and baseline (i.e., pre-intervention) measures of these variables were included as covariates in the respective analyses.

Aim One

Hypothesis 1.1: The effect of Group on attentional bias on the VP task at the laboratory assessments was examined using LMM. Because each VP assessment in the lab included new as well as old (untrained) stimuli, Picture Type (old vs. new) was

included as a within-subject variable. A significant parameter estimate for Group would reveal that, averaged over all lab assessments, the AR group exhibited lower (or higher) levels of attentional bias than the Control group. A significant parameter estimate for the Group by Visit interaction term would reveal that an effect of Group changed over time, and a significant parameter estimate for the Group by Picture Type interaction would reveal that the effect of Group differed in old and new pictures.

Hypothesis 1.2: The effect of Group on attentional bias on the smoking Stroop task at the laboratory assessments was examined using LMM. A significant parameter estimate for Group would reveal that, averaged over all lab assessments, the AR group exhibited lower (or higher) levels of attentional bias on the smoking Stroop task than the Control Group. A significant parameter estimate for the Group by Visit interaction term would reveal that an effect of Group changed over time.

Hypothesis 1.3: The effect of Group on attentional bias assessed with the visual probe task on the PDA was examined using LMM. A significant parameter estimate for Group would reveal that, averaged over all PDA assessments, the AR group exhibited lower (or higher) levels of attentional bias than the Control group. A significant parameter estimate for the Group by Day interaction term would reveal that an effect of Group changed over time, and a significant parameter estimate for the Group by Picture Type interaction would reveal that the effect of Group differed in old and new pictures.

Hypothesis 1.4: The effect of Group on self-reported attention capture by smoking cues (on the SABQ) was examined using LMM. A significant parameter estimate for Group would reveal that, averaged over all lab assessments, the AR group exhibited lower (or higher) SABQ ratings than the Control group. A significant parameter

estimate for the Group by Visit interaction term would reveal that an effect of Group changed over time.

Hypothesis 1.5: The effect of Group on self-reported attentional bias assessed on the PDA was examined using LMM. A significant parameter estimate for Group would reveal that, averaged over all PDA assessments, the AR group exhibited lower (or higher) levels of self-reported attentional bias than the Control group. A significant parameter estimate for the Group by Day interaction term would reveal that an effect of Group changed over time.

Hypothesis 1.6: The effect of Group on attentional bias measures derived from the mobile eye tracker was examined using LMM. A significant parameter estimate for Group would reveal that, averaged over all eye tracker assessments, the AR group exhibited lower (or higher) levels of attentional bias than the Control group. A significant parameter estimate for the Group by Day interaction term would reveal that an effect of Group changed over time. The primary outcome is the proportion of time spent looking at the smoking stimulus. All eye movement analyses were conducted on data from the first 5 seconds in the smoking lab.

Hypothesis 1.7: The effect of Group on self-reported exposure to smoking cues assessed on the PDA was examined using LMM. A significant parameter estimate for Group would reveal that, averaged over all PDA assessments, the AR group exhibited lower (or higher) levels of self-reported exposure to smoking cues than the Control group. A significant parameter estimate for the Group by Day interaction term would reveal that an effect of Group changed over time.

Aim Two

Hypothesis 2.1: The effect of Group on self-reported craving ratings (on the Questionnaire for Smoking Urges; QSU) was examined using LMM. A significant parameter estimate for Group would reveal that, averaged over all lab assessments, the AR group exhibited lower (or higher) QSU ratings than the Control group. A significant parameter estimate for the Group by Visit interaction term would reveal that an effect of Group changed over time.

Hypothesis 2.2: The effect of Group on self-reported cued and non-cued craving assessed on the PDA was examined using LMM. A significant parameter estimate for Group would reveal that, averaged over all PDA assessments, the AR group exhibited lower (or higher) levels of self-reported craving than the Control group. A significant parameter estimate for the Group by Day interaction term would reveal that an effect of Group changed over time. Non-cued and cued craving was tested in separate models.

Aim Three

Hypothesis 3.1: The effect of Group on Carbon Monoxide (CO) levels was examined using LMM. A significant parameter estimate for Group would reveal that, averaged over all lab assessments, the AR group exhibited lower (or higher) CO levels than the Control group. A significant parameter estimate for the Group by Visit interaction term would reveal that an effect of Group changed over time.

Hypothesis 3.2: The effect of Group on salivary cotinine levels was examined using LMM. A significant parameter estimate for Group would reveal that, averaged over all lab assessments, the AR group exhibited lower (or higher) cotinine levels than the

Control group. A significant parameter estimate for the Group by Visit interaction term would reveal that an effect of Group changed over time.

Hypothesis 3.3: The effect of Group on cigarettes smoked per day (assessed in a smoking diary or on the PDA) was examined using LMM. A significant parameter estimate for Group would reveal that the AR group reported lower (or higher) levels of smoking than the Control group. A significant parameter estimate for the Group by Day interaction term would reveal that an effect of Group changed over time.

Power Analyses

Power analyses were conducted for primary hypotheses 1.3 and 2.2 for EMA data. Power estimates accounted for the fact that repeated observations from the same person will be correlated, indexed by the intraclass correlation coefficient (ICC). It was assumed that participants would complete 75% of the PDA field assessments, and *sample sizes* would equal 30, 26, and 22 per group at visits 1, 2, and 3, respectively (504 VP assessments in total completed on the PDA). If the ICC = .1 (or .3), then the effective sample size = 258 (131), and power = .98 (.81) to detect an effect size of Cohen's $f = 0.25$ (a medium effect size, equivalent to $d = 0.50$) for the main effect of Group on attentional bias. Craving is assessed at every assessment and so power would be greater for the same ICCs. Kerst and Waters (109) reported effect sizes of $d = 0.69$ (a large-to-medium effect size) and $d = 0.51$ (medium effect size) for the effect of 1-week AR on attentional bias and cued craving, respectively. These data bolster confidence that the study is adequately powered to detect main effects of Group on attentional bias and craving assessed in the field. For lab assessments, power was lower due to the smaller number of assessments (2 groups x 48 assessments per group (estimated) = 96 assessments). If the ICC = .1 (or .3),

then the effective sample size = 89 (76), and power = .65 (.58) to detect an effect size of Cohen's $f = 0.25$ (a medium effect size, equivalent to $d = 0.50$) for the main effect of Group on a dependent variable. If the effect size is large in the population (Cohen's $f = 0.40$), then power = .96 (.93) to detect an effect of Group.

Validation of Group Assignment

After the study was completed, participants' group assignment was verified. The files loaded into each PDA were cross-checked with the dates the participants attended and the group assignment indicated in the researcher's records. As noted in Figure 9, one participant's condition could not be verified because he provided no PDA data (i.e., the participant provided a PDA with data erased) and the researcher could not identify his condition from records. This participant was excluded from all analyses except analysis of baseline data. This participant was included in the baseline analysis to be consistent with an intent-to-treat approach.

CHAPTER 3: Results

Descriptive Statistics

Sixty-four participants were enrolled in the study. Descriptive statistics for participants are presented in Table 7. AR and Control participants did not differ by age, sex, or race. AR and Control participants did not differ on the SABQ or QSU. The groups also had no differences on the smoking history items, including the FTND, WISDM, cigarettes per day, age of smoking initiation, and lifetime quit attempts (see Table 7). Regarding geographic location, 39.68% of participants resided in Montgomery County, Maryland; 11.11% in Prince Georges County, Maryland; 1.58% in Baltimore City, Maryland; 1.58% in Arlington, Virginia; 1.58% in Fairfax, Virginia; 1.58% in Southwest Washington, District of Columbia; 9.52% in Southeast Washington, District of Columbia.; 11.11% in Northeast Washington, District of Columbia; and 19.04% in Washington, Northwest District of Columbia.

Completers vs. Non-completers

Of the 63 participants included in the primary analyses (one participant was excluded, see 'Validation of Group Assignment'), 49 completed the study. Completion was defined as providing data at visit 3. Comparisons between Completers (n = 49) and Non-completers (n = 14) are presented in Table 8. Completers (vs. Non-completers) did not differ by age, sex, race, the SABQ, WISDM, cigarettes per day, age of smoking initiation, or lifetime quit attempts (see Table 8). Group (AR vs. Control) was not associated with Completion, $\chi^2(1) = 1.31$, $p = .25$.

EMA Descriptive Statistics

Summary statistics on dependent variables by group and day are presented in Table 10. Summary statistics by group and picture type are presented in Table 11. Across all participants who provided EMA data (n = 56), participants provided data from at least one item from 2,419 trainings and assessment. Participants completed 2,211 (91.40%) of these trainings and assessment. The AR and control group provided data from a comparable number of trainings and assessment (1,167 in the AR group and 1, 252 in the control group). They also completed a similar percentage of trainings and assessment (92.46% in the AR group and 90.42% in the control group).

` The time of day that trainings and assessments were completed was similar between the AR group (before 12 p.m.: 28.36%; 12 p.m. to 4 p.m.: 27.53%; 4 to 8 p.m.: 26.97%; and after 8 p.m.: 17.15%) and the control group (before 12 p.m.: 22.7%; 12 p.m. to 4 p.m.: 30.65%; 4 to 8 p.m.: 30.21%; and after 8 p.m.: 16.43 %).

Trainings and assessments were either random assessments (RAs; "PDA-initiated"; 74.76% of trainings and assessment) or participant-initiated (25.24% of trainings and assessment). For the AR group, 74.98% were PDA-initiated and 25.02% were participant-initiated. For the control group, 74.56 % were PDA-initiated and 25.44% were participant-initiated.

For the AR group the mean duration of completed trainings was 10.58 minutes (SD = 0.90) and 6.25 minutes (SD = 0.68) for completed assessments. For the control group the mean duration of completed trainings was 10.68 minutes (SD = 0.82) and 7.44 minutes (SD = 1.98) for completed assessments. The larger number of trials on the VP task for the trainings (vs. assessments) contributed to the difference in durations between trainings and assessments.

Participants (n = 56) contributed a mean number of 13.09 days (SD = 3.79) with PDA data. AR and Controls had a similar number of days with PDA data (AR 12.86 days (SD =3.70); Control 13.32 days (SD =3.94)), F (1, 54) = 0.21, p = .65.

Number of Trainings

Participants in the AR condition (n = 28) completed a mean of 29.07 (SD = 12.48) training tasks. Control participants (n = 28) completed a mean of 30.61 control training tasks (SD = 13.07). The two groups did not differ in the number of training tasks completed, F (1, 54) = 0.20, p = .65. Participants in the AR condition (n = 28) completed a mean of 9.46 (SD = 4.53) assessment tasks. Control participants (n = 28) completed a mean of 9.82 assessment tasks (SD = 4.57). The two groups did not differ in the number of assessments completed, F (1, 54) = 0.09, p = .77.

Specific Aim 1: Effect of AR on Attentional Bias

Hypothesis 1.1 predicted that AR (vs. Control) would decrease attentional bias to smoking-related stimuli measured by the visual probe task in the laboratory. LMM analyses on laboratory data revealed a significant main effect of Group (Table 12 and Figure 10). Aggregated over visits (and picture type), attentional bias was 19.24 ms lower in the AR group than the Control group. The Group by Visit interaction was not significant, indicating that the effect of Group did not get significantly greater over time. To examine whether the effect of Group on attentional bias was different for old vs. new pictures, the Group by Picture Type interaction term was tested (not shown in Table 12). The Group by Picture Type interaction was not significant, F (1, 125) = 1.46, p = .23). Therefore there is no evidence that the effect of Group was greater in old (vs. new) pictures. Overall, Hypothesis 1.1 was partially supported.

Hypothesis 1.2 predicted that AR (vs. Control) would decrease attentional bias to smoking-related stimuli measured by the smoking Stroop task in the lab. LMM analyses on laboratory data revealed non-significant main effects of Group, as well as a non-significant Group by Visit interaction (See Table 12). Hypothesis 1.2 was not supported.

Hypothesis 1.3 predicted that AR (vs. Control) would decrease attentional bias to smoking-related stimuli measured by the visual probe task in the field. LMM analyses of field data revealed a significant main effect of Group in the field. The Group by Day interaction term was not significant (Table 13 and Figure 11). Aggregated over assessments (and picture type), attentional bias was 24.06 ms less in the AR group than in the Control group. To examine whether the effect of Group on attentional bias was different for old vs. new pictures, the Group by Picture Type interaction term was tested (not shown in Table 13). The Group by Picture Type interaction was not significant, $F(1, 372) = 0.22$, $p = .64$. Therefore there is no evidence that the effect of Group was greater in old (vs. new) pictures. Overall, Hypothesis 1.3 was partially supported.

Hypothesis 1.4 predicted that AR (vs. Control) would report lower SABQ ratings in the laboratory. LMM analyses on laboratory data revealed a non-significant main effect of Group, as well as a non-significant Group by Visit interaction (See Table 12). Hypothesis 1.4 was not supported.

Hypothesis 1.5 predicted that AR (vs. Control) would report lower ratings of self-reported attentional bias assessed in the field. LMM analyses of field data revealed a non-significant main effect of Group in the field, as well as a non-significant Group by Day interaction (See Table 12 and 13). Hypothesis 1.5 was not supported.

Hypothesis 1.6 predicted that AR (vs. Control) would exhibit lower attentional bias on the mobile eye tracker in the laboratory. LMM analyses on laboratory data revealed a non-significant main effect of Group, as well as a non-significant Group by Visit interaction (See Table 12). Hypothesis 1.6 was not supported.

Hypothesis 1.7 predicted that AR (vs. Control) would exhibit lower levels of self-reported exposure to smoking cues assessed in the field. LMM analyses on field data revealed a non-significant main effect of Group, as well as a non-significant Group by Day interaction (See Table 13). Hypothesis 1.7 was not supported.

Specific Aim 2: Effect of AR on Craving

Hypothesis 2.1 predicted that AR (vs. Control) would exhibit lower levels of craving assessed on the QSU in the laboratory. LMM analyses on laboratory data revealed a non-significant main effect of Group, as well as a non-significant Group by Visit interaction (See Table 12). Hypothesis 2.1 was not supported.

Hypothesis 2.2 predicted that AR (vs. Control) would report lower levels of cued and non-cued craving in the field. LMM analyses on field data revealed non-significant main effects of Group, as well as a non-significant Group by Day interaction (See Table 13 and Figure 12). Data on cued and non-cued craving were also collected in the laboratory. LMM analyses on laboratory data revealed a non-significant main effect of Group, as well as a non-significant Group by Visit interaction (See Table 12). Hypothesis 2.2 was not supported.

Specific Aim 3: Effect of AR on Smoking

Hypothesis 3.1 predicted that AR (vs. Control) would have lower CO levels assessed in the laboratory. LMM analyses on laboratory data revealed a non-significant

main effect of Group, as well as a non-significant Group by Visit interaction (See Table 12). Hypothesis 3.1 was not supported.

Hypothesis 3.2 predicted that AR (vs. Control) would have lower cotinine levels assessed in the laboratory. LMM analyses on laboratory data revealed a non-significant main effect of Group, as well as a non-significant Group by Visit interaction (See Table 12). Hypothesis 3.2 was not supported.

Hypothesis 3.3 predicted AR (vs. Control) would report smoking fewer cigarettes on the smoking diary assessed in the field. LMM analyses on field data revealed a non-significant main effect of Group, as well as a non-significant Group by Visit interaction (See Table 12). Cigarette smoking on the PDA in the field was assessed with the 5-point item described earlier. LMM analyses on field data revealed no main effect of Group, but did reveal a significant Group by Day interaction (See Table 13 and Figure 13). Follow up analyses revealed that smoking rate assessed in the field declined over days in the AR group, PE = -0.04, SE = 0.01, F (1, 26) = 10.95, p = .003, but not in the Control group, PE = 0.00, SE = 0.02, F (1, 27) = 0.02, p = .89. Hypothesis 3.3 was partially supported (PE= Parameter Estimate, SE = Standard Error).

Supplementary Analyses

Supplementary Analyses on Effect of AR on Attentional Bias

Effect of AR on Attentional Bias on New Pictures

As noted earlier, the effect of AR on attentional bias was not moderated by Picture Type (i.e., there was no Group by Picture Type interaction) for attentional bias assessed in the lab and field. Therefore the effect of AR was not different on old vs. new pictures. Here the effect of AR on new pictures was assessed. Table 11 reports attentional

bias on new pictures during EMA and in the lab for "Week 1" (EMA Week 1, lab visit 2) and "Week 2" (EMA Week 2, lab visit 3). In all conditions the mean attentional bias of Control participants had a positive sign, whereas the mean attentional bias of AR participants had a negative sign. This finding suggests that AR generalizes to new pictures. The effect of AR on new pictures was formally tested in a LMM which included Group (AR vs. Control), Setting (EMA vs. Lab), Week (Week 1 vs. Week 2), and baseline attentional bias as covariate, examined the effect of Group on attentional bias on new pictures. To maximize power, this analysis included the EMA and lab data in a single analysis. This analysis revealed a main effect of Group, F (1, 91) = 4.55, PE = 27.67, SE = 12.97, p = .036, thereby bolstering the case that AR reduced attentional bias on new pictures.

Effect Size for Effect of AR

The parameter estimates shown in Tables 12 and 13 are unstandardized measures of effect size. The between-group difference is expressed in terms of ms, the units of the attentional bias variable. A standardized effect size measure for the main effect of Group on attentional bias assessed in the field was computed by calculating subject means (over all assessments) and computing a regression in which mean attentional bias was the dependent variable, Group was the independent variable, and attentional bias at baseline was the covariate. The t statistic from the regression was converted to a Cohen's d value using the formula Cohen's d = $2t/\sqrt{df}$. This yielded a Cohen's d of 0.60 for the main effect of Group on attentional bias (when controlling for baseline attentional bias). A Cohen's d of 0.60 is considered a medium-to-large effect size. Using the same formula applied to the lab data, a Cohen's d of 0.80 was obtained, a large effect size.

Attentional Bias on Control Trainings

An attentional bias score can also be computed on Control trainings, because on control trainings the probe is equally likely to replace smoking and neutral pictures. (Attentional bias scores cannot be computed on AR trainings because the probe never replaces smoking pictures). Control participants exhibited a significant attentional bias (i.e., an attentional bias significantly different from 0) on Control trainings, *PE* = 7.31, *SE* = 3.34, *p* = .037, which was not significantly different (*p* = .18) from attentional bias assessed at Assessments, *PE* = 14.88, *SE* = 6.85, *p* = .039. In sum, in the field Control participants exhibited a significant (positive) attentional bias at both Assessments and Trainings. In contrast, at Assessments the AR participants exhibited a (non-significant) *negative* attentional bias, *PE* = -8.80, *SE* = 7.10, *p* = .22. That is, AR participants tended to exhibit a negative attentional bias (avoidance, faster responses to probes that replaced neutral pictures), but this bias was not significantly different from zero. However, at lab visit 3 only, AR participants did exhibit significant avoidance (p = .03). This finding suggests that the Control group consistently exhibit a bias toward smoking cues and the AR group tend to have a bias away from smoking cues.

Sensitivity Analyses

Sensitivity analyses were conducted to evaluate the robustness of reported findings for attentional bias. As noted earlier, the primary analyses were conducted on data on assessments on which participants reported no more than one interruption. The results presented in Tables 12 and 13 did not change if all assessments were used in analyses. As noted in Table 7, SABQ scores were (non-significantly) higher at baseline in the Control group than the AR group. All analyses were recomputed including SABQ

baseline scores as a covariate. Inclusion of baseline SABQ scores did not change the significance of any of the reported findings. Inclusion of the FTND, a covariate included in Kerst and Waters (109), also did not change the significance of any of the main findings. Finally, due to researcher error, seven control participants were provided control training/assessment files that were different from those they were scheduled to receive. Analyses were conducted to determine if these seven participants had outcomes that were different to those of the other control participants. No effect was found.

Post-hoc Analyses

Examination of Figure 11 appears to indicate that attentional bias unexpectedly increased from day 12 to day 13 in the AR group. However, there was no evidence that this increase was statistically significant. A post-hoc LMM comparing attentional bias data on day 12 and day 13 did not reveal a significant difference in an analysis using an uncorrected *p* value ($p = .13$).

Table 9 and Figure 10 appear to indicate that attentional bias increased in the control group at lab sessions 2 and 3, compared to baseline. However, there was no evidence that the apparent increase was statistically significant. A post-hoc LMM comparing attentional bias data at visit 2 and baseline did not reveal a significant difference in an analysis using an uncorrected *p* value ($p = .06$), and the same was true for the comparison of attentional bias at baseline and visit 3 ($p = .21$).

Assessment of Advertisements

Although this study did not find an effect on advertisements, the data on daily exposure to advertisements may inform future interventions. Control participants reported seeing 0 advertisements (since the previous assessment) on 66.75% of PDA field

assessments, one advertisement on 12.15% of PDA field assessments, two advertisements on 11.08% of PDA field assessments, three advertisements on 5.67% of PDA field assessments, and four advertisements on 4.31% PDA field assessments. AR participants report seeing 0 advertisements on 66.13% of PDA field assessments, one advertisements on 9.95% of PDA field assessments, two advertisements on 8.51% of PDA field assessments, three advertisements on 6.09% PDA field assessments, and four or more advertisements on 9.32% PDA field assessments. Therefore, participants reported seeing at least one advertisement on about one third of PDA field assessments. The mean number of advertisements seen each day is in Table 10. These findings indicate that participants are frequently exposed to advertisements. However future research on AR and advertisements is needed because AR did not affect advertisement exposure in the current study.

Supplementary Analyses on Effect of AR on Craving

Figure 12 indicates the cued craving appeared to decline over time in both groups. Although not a primary hypothesis, given the results of Enock and colleagues (52) the effect of Day was examined. The effect of Day was significant both for the cued craving item, F (1, 54) = 7.12, p = .01, and for the non-cued craving item, F (1, 54) = 5.30, p = .03. As noted earlier, the Group by Day interaction was not significant, meaning that the declines over time were not different in the two groups. In the laboratory sessions, craving also declined over sessions on the cued craving item, F (2, 95) = 8.08, $p < .001$, and the non-cued craving item, F (2, 95) = 4.88, p = .01. Therefore, craving does generally decline over time.

As noted earlier, the main effect of Group was not significant on the cued craving item (p = .11). The parameter estimate (95% confidence intervals) was 0.45 (-0.11, 1.01). Using the methods described earlier, Cohen's d for the effect of Group, when controlling for baseline cued craving, was 0.20, a small effect size. Recall that Kerst (108)reported a main effect of Group. In Kerst (108), the parameter estimate (95% confidence intervals) was 0.77 (0.00. 1.55). As can be seen, the confidence intervals from the current study overlap substantially with those of Kerst (2013), meaning that the observed data for craving in the current study were not significantly different from the findings of Kerst (2013). Also consistent with Kerst (2013), ratings on the cued craving item were not higher than ratings on the non-cued craving item (p = .54).

Supplementary Analyses on Effect of AR on Smoking

Effect of AR on PDA smoking

To assess the robustness of the Group by Day interaction for reported smoking on the PDA, analyses were conducted treating the variable as an ordinal dependent variable using SAS PROC GLIMMIX (SAS PROC MIXED assumes the dependent variable is an interval-level continuous variable that is normally distributed in the population). This analysis also revealed a significant Group by Day interaction. Follow up analyses again revealed that smoking rate assessed in the field declined over days in the AR group, PE = 0.07, SE = 0.02, F (1, 26) = 9.18, p = .005, but not in the Control group, PE = 0.00, SE = 0.03, F (1, 27) = 0.01, p = .92.

Other Supplementary Analyses

Heaviness of Smoking

Thirty-three "heavy" (11+ cigarettes per day) and 31 "light" (5-10 cigarettes per day) smokers were recruited into the study. There was no difference in the number of heavy and light participants recruited into the AR (Heavy: 15, Light: 16) vs. Control (Heavy: 18, Light: 15) group, χ^2 (1) = 0.24, p = .62. As expected, participants identifying as heavy smokers had higher salivary cotinine levels at baseline, t(57.11) = 3.62, p = .0006, and higher CO levels at baseline, t(62) = 2.21, p = .03. A detailed analysis of heaviness of smoking is beyond the scope of the dissertation. Here, the author reported whether the observed significant effects of Group are moderated by heaviness of smoking. For attentional bias assessed in the laboratory, there was no evidence that the effect of Group differed in Heavy vs. Light smokers, i.e., no Group by Heaviness interaction: F (1, 126) = 0.16, PE = 5.36, SE = 13.14 p = .69. The same was true for attentional bias measured in the field; Group by Heaviness interaction: F (1, 374) = 0.03, PE = -3.77, SE = 0.86, p = .85. Similarly, there was no evidence that the observed Group by Day interaction on smoking in the field was moderated by Heaviness, i.e., no Day x Group by Heaviness interaction, F (1, 2057) = 0.38, PE = 0.03, SE = 0.04, p = .54. Future analyses can examine whether other measures of heaviness of smoking, such as cotinine levels, moderate the effect of AR on outcomes.

Assessment Type

As noted earlier, trainings and assessments were delivered at either random assessments (RAs; 74.76% of trainings and assessment) or "participant-initiated" assessments (25.24% of trainings and assessment). As noted earlier, Assessment Type

(RAs vs. participant-initiated) was entered as a covariate in all EMA analyses. Analyses explored whether the observed significant effects of Group were moderated by Assessment Type. For attentional bias, the Group by Assessment Type interaction was not significant, F (1, 371) = 0.22, PE = -10.10, SE = 21.47, p = .64. Similarly, for smoking rate the Group by Day by Assessment Type interaction was not significant, F (1, 2054) = 0.04, PE = 0.01, SE = 0.03, p = .85. In sum, there was no evidence that the effects of Group differed by Assessment Type. Future analyses can examine whether the effect of Group on other outcome variables is moderated by Assessment Type.

Post-Treatment Questionnaire

The Post-treatment questionnaire assessed blinding to condition, the acceptability of treatment, and intent to quit in the next 30 days. For the item that assessed blinding, 65.4% of AR participants reported that they were in the active group and 77.3% of Control participants reported they were in the active group. In other words, participants tended to believe they were in the active group whether they were in the AR or Control group.

Regarding acceptability of treatment, 88.5% of AR participants and 86.4% of the Control participants rated the treatment as acceptable. The item that assessed recommending the treatment to a friend was on a scale of 0 to 4 with the anchors "definitely not" to "definitely." There was no significant difference between groups on this item, t(46) = -0.95, p = .35 (AR: M = 3.35 , SD = 0.85 ; Control: M = 3.09 , SD = 1.02). The item that assessed if participants believed that the intervention was interesting or boring was on a scale of 0 to 4 with the anchors "very boring" to "very interesting." There was no significant difference between groups on this item, t(46) = -1.89, p = .07

(AR: *M* = 2.65 , *SD* = 1.13; Control: *M* = 2.00 , *SD* = 1.27). The item that assessed intent to quit was on a scale of 0 to 4 with the anchors “definitely not” to “definitely.” There was no significant difference between groups on this item, *t*(46) = 1.23 *p* = .22 (AR: *M* = 1.69, *SD* = 1.01; Control: *M* = 2.09, *SD* = 1.23).

CHAPTER 4: Discussion

The current doctoral dissertation study examined the efficacy of AR administered on a mobile-device in a sample of African American smokers. Participants (N = 64) were randomly assigned to an AR or Control training condition. Participants were given a mobile-device for 2 weeks, which prompted them to complete up to three AR (or control) trainings per day. Participants completed assessments of attentional bias, craving, and smoking both in the lab and in the field (one time per day). The primary aims were to assess the effect of AR on attentional bias, craving, and smoking

The main findings of this study were as follows. First, aggregated over all assessments, AR (vs. Control training) significantly reduced attentional bias to smoking cues on the VP task both in the laboratory and in the field. The effect of AR did generalize to new pictures on the VP task, but did not generalize to attentional bias assessed on the Smoking Stroop task, or to self-report measures of attentional bias. Second, AR (vs. Control training) did not reduce craving. Third, AR, but not Control training, significantly reduced the number of cigarettes smoked over time, when assessed on the PDA. There was no effect of AR on biological measures of smoking, or smoking reported on the smoking diary. These findings are discussed in more detail below.

Effect of AR on Attentional Bias

Aggregated over all assessments, the AR group exhibited a significantly reduced attentional bias in the lab and field on the VP task. There was consistent evidence that Control participants, but not AR participants, exhibited attentional bias. For example, the Control group exhibited a significant attentional bias on PDA field assessments both at assessments and at control trainings (from which attentional bias scores can be

computed). In contrast, the AR participants tended to exhibit a negative attentional bias (attentional bias away from smoking cues, or "avoidance") although this bias was not significantly different from zero in the field data. This outcome is similar to the data reported by Kerst and Waters (109) who also found a between-group difference in attentional bias after day 5, but the AR group did not exhibit significant avoidance. It therefore appears to be difficult to produce significant avoidance in the AR group.

This point notwithstanding, the current study did find significant avoidance at lab visit 3, the final assessment of the study. This finding is similar to the results of a multi-session laboratory AR protocol for smokers (132). However`, not all laboratory AR smoking studies have demonstrated avoidance in the AR group (see Table 3) (11; 145). Notably, these studies involved a single session of AR which suggests that multiple sessions (in the lab or on a PDA) may be necessary to produce avoidance. Overall, there is modest evidence that AR can produce avoidance in smokers, although avoidance may be a desired outcome (171). It should be noted that even if avoidance is not achieved, the reduction of attentional bias in the AR group compared to Controls may be an important clinical change.

Although Figure 10 appears to indicate that the effect of AR became larger over time, the Group by Time interaction terms were not significant either in the lab (No Group by Visit interaction) or field (no Group by Day interaction). As indicated in Figure 11, the attentional bias of the AR group appeared to increase on days 13 and 14, although this finding did not reach statistical significance. If real, the reason for such an increase is not clear. It is possible that participants, including AR participants, responded differently during days 13 and 14 because they perceived the study to be nearing completion and

therefore they became less engaged in completion of the cognitive assessments. It should also be noted that a different pattern of data was observed in the lab at visit 3 (as noted above). Kerst and Waters (109) did obtain a Group by Day interaction, but this study used a mixed-race sample (61.7% African American) and different inclusion criteria with respect to smoking rate (10+ cigarettes per day). In addition, the current data also showed a non-significant pattern consistent with a Group by Time interaction (Figure 10). Overall, more training may be required to observe a Group by Day interaction in African American smokers.

Interestingly, the effect of AR generalized to new pictures in both the laboratory and field. This finding is consistent with some (132) but not all previous research (65). One methodological factor that may explain the discrepant findings is the number of training sessions (see Table 3). Note that Field (65) only included a single training session. It is possible that multiple training sessions are necessary for the effect of AR to generalize to new pictures.

In the current study, neither group showed an attentional bias toward smoking cues at baseline. This finding is inconsistent with previous laboratory studies (11; 65; 132) and the field (109). Attentional bias may be less robust in the current sample because it included a mix of light and heavy smokers. Attentional bias in light smokers has not been well-studied but it is possible that attentional bias is less robust in light smokers because they are less dependent (163). In addition, in the Control group attentional bias at baseline was not significantly different from attentional bias at session 2 or session 3.

While there was an effect of AR on attentional bias assessed on the VP task, the current study did not demonstrate an effect of AR on any other measures of attentional bias. The results in this study are consistent with previous studies that have found no effect of AR on the modified Stroop task (64; 65). Although the VP and modified Stroop are both reaction time measures of attentional bias, the modified Stroop involves distinct cognitive processes from the VP. There is empirical evidence that attentional bias assessed by the VP task and the modified Stroop task do not correlate (150). Theoretically, there is reason to believe that the two tasks assess different aspects of attention. In the context of Posner's attention systems framework, the VP task appears to map on to the orienting network, whereas the Stroop task maps on to the executive network (178). Posner describes orienting as the selection of information for sensory input. This description is consistent with VP task in which participants orient to smoking vs. neutral stimuli. In contrast, the executive network involves monitoring and resolving conflict among thoughts, feelings, and responses. These processes are required for the Stroop task where participants are required to respond by pressing the color of words that are emotionally salient and may cause distraction. Also, there is fMRI evidence suggesting that the modified Stroop increases activity in the dorsolateral frontal lobe, a region associated with executive control (40).

Therefore, it is possible that another type of AR intervention may be necessary to target the attentional bias assessed by the modified Stroop task. For instance, Matthews and MacLeod (136) developed such an intervention in the domain of anxiety. Participants were presented with a series of colored words which were either threat words or neutral words. In the "active" group participants, were always required to make a grammar

judgment about neutral words and a color-naming response to threat words. In the control group, participants were always required to make a grammar judgment about threat words and a color-naming response to neutral words. The grammar judgment trials required participants to access the meaning of words and the color naming trials allowed participants to ignore the meaning of the word. The idea is that, over time, participants in the active group implicitly learn to attend to surface features of threatening stimuli and semantic features of neutral stimuli. Stated simply, over time participants in the active group should implicitly learn to process neutral stimuli more deeply than threatening stimuli. After training, participants in the active group exhibited less attentional bias to threat in a modified Stroop task than those in the control group.

In summary, the results suggest that the effect of AR administered by the VP task is task specific and does not reduce all aspects of attentional bias. Fadardi and Cox (56) developed an AR task (AACPT: Alcohol Attention-Control Training Program) that was designed to reduce attentional bias assessed by the modified Stroop task. They assessed the effect of the AACPT on attentional bias to alcohol assessed by the modified Stroop task and on alcohol use. As part the AACPT, participants were required to color-name the outline of a non-alcoholic container but to ignore an alcoholic container presented simultaneously on the screen. In addition, participants were given feedback as to their level of distraction by alcohol cues and encouraged to try to reduce the influence of alcohol cues. Over repeated trainings, attentional bias assessed by the modified Stroop task declined over time. In addition, alcohol use in harmful drinkers declined over time. One limitation of this study is that there was no control group. If these findings were replicated and extended with the use of a control group, then the AACPT task described

by Fadardi and Cox (56) may be a useful way to reduce attentional bias assessed by the modified Stroop task.

An effect of AR on the subjective attentional bias measures (SABQ, and 1-item PDA measure of self-reported attentional bias) was not observed, nor was an effect found on self-reported exposure to tobacco advertisements or on attentional bias assessed by the mobile eye assessment. First, it is possible that the effect of AR does not generalize to self-reported exposure to tobacco advertisements because these stimuli were not included in the training. Although, as noted above, the effect of AR did generalize to new pictures, the new pictures were more similar in content to the old pictures than the tobacco advertisements. Fortunately, AR interventions can be easily modified and it is possible to use pictures of tobacco advertisements and Point of Sale environments as training stimuli. Second, the findings from the mobile eye assessment are not conclusive because the analysis was limited to the first 5 seconds. Theoretically, the first 5 seconds is of greatest interest because they capture automatic attention to smoking cues. But it is possible that AR has an effect on the amount of time spent looking at smoking stimuli after the first 5 seconds. Indeed, Kerst (108) found that AR decreased attention to cues assessed by the mobile eye over a 1-minute period but not in the first 5 seconds. This analysis was beyond the scope of the present dissertation study but future analyses will explore the effect of AR on the mobile eye assessment over the 1-minute period. Last, the absence of an effect of AR may be influenced by the fact that smokers in this study did not wish to quit. The effect of AR on self-reported attention may be easier to detect in smokers who are trying to quit and therefore trying to avoid smoking cues than in non-treatment-seeking smokers.

Taken together these findings suggest that AR can reduce attentional bias in a sample of light and heavy African American smokers assessed by the VP task but not by other attentional bias assessments. While this finding is promising, much more work is needed to determine if the effect of AR can generalize to other attentional bias assessments or whether additional retraining tasks and/or more trainings are required. In particular, more work is needed to determine if AR reduces attention to real-world smoking cues.

Effect of AR on Craving

Compared to control training, AR did not reduce craving in the current study. This finding was true for both cued and non-cued craving assessed in the lab and field, as well as Questionnaire for Smoking Urges (QSU) scores assessed in the lab. For cued craving, this finding was counter to hypotheses and different from previous findings (109). However, Figure 12 revealed that cued craving tended to be (non-significantly) lower in the AR group than in the Control group, and supplementary analyses revealed that the parameter estimates were not markedly different in the two studies. Another issue with the cued craving data was that the craving on the cued craving item was not significantly greater than that on the non-cued craving item. This finding is not easy to interpret because, as noted earlier, the cued craving item was always presented first. However, if it were true that the cued craving pictures did not elicit craving, then the ability of AR to reduce cued craving would be compromised. In addition, there is no way to verify whether participants actually looked at the pictures. Further research is required to determine the effect of AR on cued craving.

Overall, there is currently little evidence that AR reduces non-cued craving in the lab with several studies reporting null findings (65; 109; 132; 199). The current study adds to the evidence that AR does not influence non-cued craving. If AR does influence behavior, then it is unlikely to be mediated by non-cued craving.

An incidental finding of the current study was that cued craving ratings (and non-cued craving ratings) declined over time. This decline was not significantly different between the two groups. Enock and colleagues (52), in a multi-session smartphone-based AR study, similarly reported a decline in social anxiety ratings over time in both an active AR and Control training group. The reason for such a decline in the current study is not clear but could be due to demand characteristics. Alternatively both AR and control training may have served as an "exposure" manipulation whereby the presence of smoking cues (during VP tasks) is decoupled from smoking. A control group, such as a waitlist control group (52) or a group that practices another cognitive task (199), could be used to determine if the effect on craving is real. For example, if participants in a waitlist control group reported no change in craving over time and participants receiving both AR and control training reported a decline in craving (as was the case in the current study), then both types of training may have the potential to decrease craving.

Effect of AR on Smoking

The AR group, but not the control group, reported smoking significantly fewer cigarettes over time on the PDA item that assessed smoking since the last assessment. However, there was no effect of AR on biological measures of smoking, or reported cigarettes smoked on the smoking diary. The effect of AR may be more apparent on the PDA smoking item than the smoking diary because it assesses smoking more frequently

and with less opportunity for recall bias. Reported smoking on smoking diaries are subject to a number of biases and subject non-compliance (218). In contrast, the PDA item is completed in real-time. In addition, breath CO would be expected to assess recent smoking over the past day, and cotinine would largely assess smoking over the past two days, meaning that the assessment of smoking is less comprehensive than with the PDA smoking item.

It is also possible that smokers in the AR group smoked fewer cigarettes per day but continued to take in the same amount of nicotine because they smoked those cigarettes more intensely. This phenomenon is referred to as compensatory smoking (17). Smokers can compensate by inhaling more deeply, by taking more puffs on each cigarette, and by blocking ventilation holes (17). If smokers in the AR group engaged in this practice then they may exhibit no change in biological, measures of smoke exposure despite smoking fewer cigarettes per day.

A limitation with the data from the PDA item is that, although smoking declined over time in the AR group, the smoking rate of the AR group was not lower than the smoking rate of the Control group at the end of the study (Figure 13). In addition, a significant effect of AR was not observed on this item in the laboratory. Furthermore, previous research has not demonstrated an effect of AR on non-treatment smokers (109; 132) suggesting that any effect of AR on smoking in this population is likely to be modest. Notably, the current study included more training sessions than the previous studies, which suggests that more training may be necessary to produce an effect of AR on smoking.

Limitations

There were a number of limitations to this study. First, although the majority (87.5%) of the participants were able to provide data in the field, there were some surprising difficulties with data collection. Two participants (3.2%) reported that they lost the PDA during Week 1 and subsequently dropped out. Another two participants (3.2%) reported that they lost the PDA during Week 2 (these participants did provide data during Week 1). Six additional participants (9.4%) provided no field data in the study due to letting the battery die (in which case all data are lost), damaging the PDA, or PDA error. Future studies can address these concerns by utilizing smartphones that transmit the data in real time, meaning that no data are lost if the battery dies or the device is lost. Speculatively, the most conscientious or reliable participants contributed the most data to the study. If so, then participant attrition due to participant error may reduce the external validity of study findings. However, comparisons between Completers and Non-completers yielded no significant difference at baseline. In addition, participant attrition could lead to subtle differences in the characteristics of the individuals in the two groups, thereby potentially undermining the internal validity of the study. Intent-to-treat (ITT) analyses can be used to partially mitigate this problem. Such analyses were beyond the scope of the current project.

Second, as noted earlier, the study sample was non-treatment seeking. It is not known how AR may influence individuals trying to quit. . Future research could also examine whether the effect of AR on study outcomes is moderated by baseline variables such as stage of change or readiness to quit.

Third, the study was not designed to compare AR tailored for African American smokers to a non-tailored AR intervention. Therefore it cannot be definitively concluded

that tailoring AR to specific groups is necessary. It will be important for future studies to examine if tailoring improves the effect of AR.

Fourth, although old pictures were defined as those on which participants were scheduled to receive training, the author cannot be certain that participants actually did receive training on those pictures prior to assessment. For example, a participant may have failed to complete any AR or control trainings prior to the assessment using those pictures. In principle, it is possible to determine those assessments on which a participant did receive at least one training prior to assessment. This analysis was beyond the scope of the current study but could be pursued in future research.

Fifth, it would have been useful to comprehensively assess participants' awareness of the relationship (or "contingencies") between dot location and picture location during the study. "Contingency awareness" is not often assessed in the AR literature, but conscious awareness of the training contingencies could potentially moderate the effect of AR on outcomes. For example, if an AR participant is aware of a contingency, he or she may have the opportunity to use conscious processes to shift attention, in addition to the automatic processes involved in AR. There is currently no consensus on whether participant awareness of contingencies would be advantageous or not (134).

Sixth, the study did not include an objective measure of attention to smoking cues in the environment. Attention to smoking cues was assessed in several ways including the SABQ, self-reported attention to cues, self-reported exposure to advertisements, and the mobile eye assessment in the laboratory. However, none of these measures can provide data on participants' actual attention to smoking cues in their daily lives. In the future it

may be possible to employ technology like Google Glasses to better capture a participant's actual exposure to cues.

Seventh, the analysis did not take into account inter-assessment intervals (i.e., the duration of time between assessments). Inter-assessment interval may explain a significant amount of variability in some of the study dependent variables. For example, the reported number of cigarettes smoked may be higher for an assessment that was completed a relatively long time after the previous assessment (because participants have more time and therefore more "opportunity" to smoke cigarettes). Future analyses can examine the effect of inter-assessment intervals.

Finally, the study used multiple dependent variables in the lab and the field, and there was no correction for multiple tests. For example, attentional bias was assessed both in the lab and field, and three measures of craving were used (two craving items on the PDA, and the QSU). Similarly there were multiple measures of subjective attention to smoking cues (e.g., SABQ and one-item PDA measures). The study also included multiple assessments of smoking including the smoking diary, PDA smoking questions, cotinine levels in saliva, and CO in breath. Therefore the type I error rate is elevated. However, for the primary outcome, attentional bias, both theory and data allowed for clear predictions to be made which somewhat obviates this concern. Nonetheless, replication of these findings is required to bolster confidence in their validity.

Strengths

The study also had strengths. First, and most importantly, this was the first study to develop a tailored AR intervention for an ethnic minority group that is known to live in cue rich environments. Second, inclusion of both light and heavy smokers is a strength

because such a sample is broadly representative of African American smokers. Third, this study comprehensively assessed attentional bias by including cognitive measures, self-reported attentional bias, mobile eye measures, and exposure to tobacco advertisements both in the lab and field.

Future Directions

Effect of Methodological Factors on AR

It is possible that the effect of AR on outcomes is diluted by the presence of assessments administered in the field. The assessments were essentially a briefer version of the control trainings, but that they included 80 (compared to 160) trials. At the end of the two week study AR participants were exposed to an average of 10 "control trainings" (assessments). "Control trainings" may diminish the effect of AR. In future studies it will be important to manipulate the proportion of assessments and AR trainings as an independent variable to examine the effect of this methodological variable.

The results of this doctoral research suggest that the next step in this line of research is to investigate the effect of AR in a sample of treatment-seeking smokers. Pairing AR with traditional smoking cessation treatments (nicotine replacement therapy and behavioral counseling) is necessary to determine if AR can have a real world impact for African American smokers.

Another important area of research is further examining the benefit of administering AR on mobile devices. The primary advantage of this approach is that participants can receive multiple trainings without visiting the laboratory. Results from this study provide evidence that individuals find several days of training acceptable. A next step in this line of research is to increase the number of weeks that participants

receive training. There is evidence from meta-analyses suggesting that the number of AR sessions moderates the effect of AR on attentional bias such that more training sessions leads to a greater effect on attentional bias. Studies that include multiple trainings had larger effect sizes (14; 83) see Halliion and Ruscio (85). In addition, the current data suggest that reported cigarette smoking decreased in a linear fashion over time. Therefore, future studies should test the effect of AR over a period of three weeks or more.

Relatedly, while most participants indicated that they would recommend the intervention to others, the monotony of the task may still be problematic. Additionally, the data for this question is confounded by "survivorship bias." It is possible that participants who found the intervention boring left the study before the post-treatment questionnaire data were collected. Fortunately, efforts have been made to "gamify" AR tasks in the anxiety literature (47). Future research should investigate a gamified AR for addiction. Moreover, the mobility of AR makes it ripe for being paired with other treatments such as nicotine replacement therapy and behavioral treatments.

Also, future AR studies may be improved by varying the stimuli type. As noted in the introduction, a strength of AR is that it can be easily modified. It is possible to train participants to attend to health-promoting behaviors. For example, researchers in the eating disorders literature have delivered AR interventions that train participants to attend away from unhealthy food and toward healthy food (105). In one study participants trained to attend toward pictures of healthy food ate more healthy foods during an experimental session compared to participants in the attend unhealthy group. This

paradigm could be applied to the smoking literature by having participants trained toward e-cigarettes, healthy foods, or other health behaviors.

To date there is little research assessing differences between AR for health-promoting stimuli versus neutral stimuli. In the anxiety literature, it has been reported that the effect size of studies training participants to attend to neutral pictures is more robust than studies training participants to attend to positive pictures (14). Studies with positive stimuli include pictures of people with smiling faces in the active condition and a control condition that is similar to the current study. However, studies directly comparing training to positive vs. control stimuli are limited in the anxiety literature and non-existent for addiction studies. Therefore, there appears to be a gap in the literature regarding which stimuli are most effective. Future studies should directly compare training toward neutral vs. health-promoting stimuli.

Including tobacco advertisements is a logical next step and would involve minor changes to the AR program. Zip code data could be used to develop stimuli specific to participants based on their zip code. The American Legacy Foundation has a database of tobacco retail storefronts and POS environments grouped by address. These pictures could be used to create a tailored set of stimuli that reflects the advertisements in their zip code. Additionally, data on marketing practices from tobacco companies can be used to select appropriate stimuli (9). While the current study took into account some marketing practices (e.g., menthol and brands targeted to African American smokers), there are likely many strategies that can be considered in future research. For example, tobacco companies may market low-income African Americans with different brands than higher income African Americans.

Relatedly, geolocation-tracking technology can used to obtain information about daily exposure to advertisements in tobacco retail outlets. Geo-location tracking involves collecting wireless global positioning system (GPS) data from participants to assess the frequency of their contacts with identified locations (e.g., a tobacco retail outlet). There is evidence that geo-location tracking can provide an objective assessment of exposure to advertisements (111). If AR proves to be a useful tool for decreasing exposure to advertisements, then a next step would be to pair AR with geo-location tracking. Geo-location tracking could be used to validate the proposed effect of AR in African American smokers (Figure 3) which is that attention to smoking cues decreases despite the environment remaining constant (assessed by geo-location tracking).

Future studies should also include a more comprehensive assessment of tobacco advertisement exposure. Specifically, collecting EMA data prior to the intervention would provide a baseline assessment of tobacco advertisement exposure. Alternatively, a laboratory self-report measure at the baseline visit could include a retrospective questionnaire about the number of advertisements seen the previous week. The former method is preferred because it is less impacted by recall bias.

Finally, there is likely heterogeneity in the number and type of advertisements advertised to African American smokers related to socioeconomic status. Thus future research should comprehensively assess socioeconomic status among African American smokers to determine the appropriate stimuli that should be used in AR.

Effect of Environment on AR

In parallel, future studies should examine the interaction between AR and the number of smoking cues in the environment. Theoretically (Figure 2), a smoker with high

attentional bias who lives in a cue-rich environment is at greatest risk of being exposed to smoking cues and would benefit most from AR. However, this interaction has not been formally tested. Unfortunately it is difficult to test this interaction without a valid objective assessment of exposure to cues. Studying AR in smokers who live in urban vs. rural neighborhoods can also help address this question. Rural neighborhoods likely have fewer tobacco retail outlets, which would suggest that there is less POS advertising. Studying smokers in rural areas could help determine to what extent the effect of AR can be observed outside of urban settings.

Moreover, there are many unknowns regarding AR and environmental cues which makes it difficult to do more than speculate about the interaction hypothesis. First, the efficacy of AR is still unknown. Therefore, it is unclear how well AR can modify attention in an environment saturated with cues. Second, tobacco advertisements are one of many cues with which a smoker may be confronted. Third, it is extremely difficult to quantify the number of proximal cues in the environment. Fourth, it is even more difficult to quantify the number of distal cues (e.g., a room in a smoker's house) and psychological cues such as mood. Last, there may be variability in the potency of cues. For example, a cigarette and lighter will likely be more potent than an advertisement.

Effect of AR on Purchasing Behaviors

As noted in the introduction AR could potentially reduce impulse purchases of cigarettes. Although purchasing behavior was not analyzed in this study, it is of interest and should be pursued in future studies. An item related to purchasing was included in this the study but was not analyzed. In addition, the assessment of purchasing behavior in this population may be more involved than originally anticipated. Many of the

participants reported to the research staff that they did not buy cigarettes in stores. Instead they purchased them as single cigarettes from other smokers and/or borrowed cigarettes from friends or family. Therefore, more formal qualitative data may be useful in order to develop a more comprehensive assessment of purchasing behavior.

Effect of AR on Comorbid Smokers

AR may also be useful for treating smokers with comorbid mental health disorders such as Post-Traumatic Stress Disorder (PTSD). Given that 40 to 86% of individuals with PTSD smoke cigarettes, the development of an AR intervention that targets both smoking and trauma cues may be useful (71). Additionally, there is emerging evidence that AR reduces PTSD symptoms. One study administered AR in conjunction with evidence-based cognitive behavioral therapies in a sample of military personnel (118). Participants in the AR group had lower PTSD symptoms and depressive symptoms at follow-up compared to the control group. Additionally, the effect of AR on trauma symptoms was mediated by change in attentional bias. Future studies should consider the development of an AR program that alternates between smoking (vs. neutral) and trauma (vs. neutral) cues.

Overall Efficacy of AR in Psychopathology

Finally, this study adds to the growing literature on the efficacy of AR. Four meta-analyses of AR have been conducted (14; 83; 85) (see table 4). The analysis conducted by Hakamata and colleagues (83), which was limited to AR for anxiety, reported a large effect of AR on attentional bias (d = 1.13) and a medium-to-large effect of AR on anxiety (d = 0.61). Hallion and Ruscio (85) examined the effect of AR on anxiety and depression and reported that AR had a small-to-medium effect on attentional

bias (g = 0.29) and a small effect on anxiety (g = 0.13) and anxiety provoked by a stressor (g = 0.28). AR did not influence depression (g = 0.06). Beard and colleagues (14)examined anxiety, depression, substance use, self-esteem, eating behaviors, and pain, and reported that - aggregated over the aforementioned conditions - the effect of AR on attentional bias was large (g = 0.79) when investigators trained participants to attend toward neutral stimuli. When investigators trained participants to attend toward neutral stimuli, the effect of AR on symptoms was a small-to-medium effect size (g = 0.36). The analysis by Mogoase and colleagues (152) examined anxiety, depression, substance use, distress in healthy participants, and eating behaviors. Aggregated over all conditions, after correcting for publication bias they reported a small-to-medium effect of AR on attentional bias (g = 0.31), and a small effect of AR on symptoms (g = 0.16).

All four meta-analyses reported a significant effect of AR on attentional bias and a significant effect of AR on symptoms. Different meta-analyses used different inclusion criteria and different statistical methodologies, which account in part for the different findings. The effect of AR on attentional bias tended to be greater than the effect of AR on symptoms. This finding is expected because the effect of AR on symptoms should be mediated through the effect on attentional bias. The effect of AR on symptoms appears to be small or small-to-medium. Interestingly, in the current study the effect size of AR on attentional bias appeared to be larger than the effect size of AR on cued craving. These findings highlight the fact that more research on AR in addiction is needed. A relatively large numbers of subjects may be required to detect an effect on symptoms (craving).

Implementation of AR to address Health Disparities

AR can potentially leverage the power of community-based research interventions (98). Community-based research emphasizes the involvement of nonacademic community members in the process of creating knowledge. For example if an African American church identified smoking as a problem for its congregation, then they could partner with academic researchers to design an intervention involving AR. In the context of community based research, members of the church would be involved with the intervention from its conception to the final product. This type of intervention is particularly powerful because the community has a perspective that is unique from academicians about how to effectively deliver an intervention. Additionally, African American churches have played a significant role in community based research interventions targeting various outcomes (8; 154). Finally, AR could be particularly beneficial in the context of community-based research because it is inexpensive, easy to use, does not require licensed clinicians, and can be used in the field.

Summary and Conclusions

The current study suggests that AR is a promising tool for reducing attentional bias in African American smokers. Multiple sessions of AR can be implemented using mobile technology in this population. There is some evidence that AR can reduce smoking behavior in a sample of adults not currently attempting to quit. The clinical utility of AR requires further investigation in the form of randomized controlled trials with African American smokers wishing to quit.

If proven to reduce smoking, then AR implemented on a smartphone may circumvent barriers to treatment including cost, medical mistrust, and physician advice

(242). AR can be widely disseminated to African Americans because of their high rates of smartphone use (160). Additionally, if developed into a smartphone application, AR can be accessible and affordable to the global population of African American smokers.

Table 1.Smoking Cessation among African Americans

Author	Population	Study Type	Analysis	Results	Notes
Trinidad et al., 2011	n = 141,603 C: 71.5% AA: 11.5% AsA/P.I : 4.5% H. :12.5%	**(S)** Tobacco Use Supplement to the Current Population Survey - Current Vs. Former Smoker	Logistic regression IV: Race DV: Cessation	Fewer AA reported being a former smoker 30% vs. 42 % ($p < .05$) **Odds of quitting for at least 6 mos. = 0.51*** for AA vs. C	
Rabius et al., 2012 (study 1)	n = 3,522 C: 85% AA: 15%	**(I)** RCT: Quit-line self-help materials vs. counseling	Chi-square IV: Race DV: Cessation: at 7 month follow-up	**No difference in Cessation** AA: 17% C: 21% $p >. 05$	
Rabius et al., 2012 (study 2)	*Louisiana,* n=4954 C: 66%, AA: 34% *Texas*,n=5209 C: 76%, AA: 24% *District of Columbia* n=1648 C: 5%, AA: 95%	**(I)** ACS Quit-line	Chi-square IV: Race DV: Cessation at 7 month follow-up	**No race difference in cessation rates** TX: 4% vs. 27%, LA: 29% vs. 27%, D.C. 23% vs. 23% $p > .05$	
Piper et al., 2010 (study 1)	n= 1,504 C: 83.9% AA: 13.6% O: 2.5%	**(I)** Conditions: Bupropi-on, nicotine lozenge; nicotine patch; nicotine patch + nicotine lozenge; bupropion + nicotine lozenge; placebo.	Logistic Regression IV: Race DV: initial cessation, 8 weeks, 6 mos. (calculated for each treatment group and combined)	**Lower cessation among AA** Initial OR = 0.34 $p < .05$ 8 weeks: 0.41, $p < .05$ 6 months = 0.59 $p < .05$	
Piper et al., 2010 (study 2)	n = 1,346 C: 87% AA: 9.5% O: 3.5%	**(I)** Conditions: Bupropion, nicotine lozenge; nicotine patch; nicotine patch + nicotine lozenge; bupropion SR + nicotine lozenge	Logistic Regression IV: Race DV: initial cessation, 8 weeks, 6 mos. (calculated for each treatment group and combined)	**No difference in cessation** $p >.05$	Combined sample (Study 1 and Study 2): **lower rates among AAs at 8** weeks w/patch + lozenge condition 28.8% vs. 52.4%; $p < .001$)
Covey et al., 2008	n=559 C: 82% AA: 5% H: 13%	**(I)** All : 8 weeks of treatment of Bupropion Nicotine Patch Counseling	Logistic Regression IV: Race DV: Abstinence 4 weeks after treatment	**Rates of cessation, lower among AA:** *OR* = 0.44 for AA, p < .05 *OR*= 0.46 for H, p < .05	

Fu et al., 2008a	n =1019 C: 33.2% AA: 29.8% Amer. In. 28.8% AsA. 8.5%	**(I)** Minnesota Health Care Programs database Use of NRT among low income smokers -7 day and 30 day abstinence	Logistic Regression IV: Race DV: Quit	**No significant race effect at 7 or 30 days.** at 7 days: C: 13.8%, AA: 13.6%, Amer. In.: 14.1, AsA 20.1%, p > .05	All participants used NRT.
Fu et al., 2008b	n = 9,216 C: 86% AA: 10% H: 3% AsA: 1%	**(S)** Collaborative Study of the Genetics of Nicotine Dependence Current vs. Former Smoker	Logistic Regression IV: Race DV: Quit, NRT	***Lower Rates among AA vs. C., OR = 0.66** $p < .05$ *Not sig. for H or AsA	The sample consisted primarily of individuals with health insurance with lower than national average rates of lifetime and current smoking.
Trinidad et al., 2005	AA, H, C. n = 61,848 to 93,5554	**(S)** The California Tobacco Surveys Years: 1990, 1993 1996, 1999, 2002	Comparisons by Age, 18-29 , 30 -45, 45+ (for each year Quit Ratios compared for all groups	**Successful cessation (+5 yrs) was lower among AA in all age groups** $p < .05$	*sample size varies by year *Greatest differences among 30-45 45+
King et al., 2004	n = 240, 488 C: 209,828 AA: 30,660	**(S)** Cross-sectional: National Health Interview Survey(1990 – 2000)	Logistic Regression IV: Race DV: Quit Proportion of Quitters	AA: 14.6 vs. W: 25.8 **Lower rates of former smokers among AA** $p < .05$	*After adjusting for covariates disparity reduced

Table 1 Note: I = Intervention; S = Survey. C = Caucasians, AA = African Americans, H = Hispanics, AsA = Asian Americans, PI = Pacific Islander, O = Other, NRT = Nicotine Replacement Therapy.

Table 2. Summary of Studies examining Attentional Bias and Relapse

Author	Population	Age	Measure	Outcome Variable	Treatment	Result	Notes
Cox et al. (2002)	14 Alcohol Abusers 20 controls (non-abusers)	Abusers 41.9 (10.6) Non Abusers 37.3(10.3)	Modified Stroop (Alcohol, Concern, Neutral) at treatment entry (T1) and prior to discharge (T2)	Relapse at 1 month follow-up	Alcohol Detox Inpatient Program	Treatment Completers vs. Non Completers (NC) vs. Controls: Group by Time $F(2,29) = 5.04$, $p < .02$, **NC had increased AB from T1 to T2.**	
Waters et al. (2003)	158 smokers	38.6 (9.5)	Modified Stroop (Smoking)	1 week abstinence	Structured behavioral cessation program	Quit Day AB predicts 1-week abstinence (OR= 1.59, CI = 1.02, 2.46, p= .04) **Lapsers had slower RT on Standard Stroop compared to non-lapsers**	RT on Standard Stroop predicted time to 1st (HR _ 1.03, CI = 1.02, 1.66, p =.03)
Carpenter et al. (2006)	80 dependent drug users in outpatient treatment study of pharmacotherapy + CBT (45 Cocaine, 25, Marijuana, 10 Heroin)	38.6 (8.1)	Modified Stroop (Cocaine, Marijuana, Heroin)	Treatment retention; drug use (days/wk); positive urine samples	Pharmacotherapy + CBT	**Pre-treatment AB associated with a cocaine positive urines (*r* =.32, p < .05)** and shorter treatment duration (*r* = -.30, p <.05)	No effect for Marijuana and Heroin
Marissen et al. (2006)	110 heroin dependent individuals (after detoxification)	34	Modified Stroop (Heroin) (pre-treatment attentional bias and post treatment)	Relapse at 3 months	Cue exposure Therapy vs. Placebo	Pre-treatment AB: *OR*= 2.23, CI= 1.06, 4.86, p = .05 **No effect for Post-treatment AB Effect found after controlling for craving**	*No biochemical verification of abstinence but participants were in a residential treatment facility

Cox et al. (2007)	158 heavy drinkers	33.6 (15.8)	Modified Stroop (alcohol) Motivation (M) Readiness to change (RTC) Family history of alcohol problems (FHAP)	Alcohol consumption	No treatment program	**Low AB had greater long-term reduction** (6 mo.) than high AAB $F(1,125)=6.224$, $p=.01$)	AB x M for short-term & long-term $F(1,125)=4.163$, $p=.04$);$F(1,125)= 4.058$, $p = .05$) AB x M x FHAP $F(1,125) =5.111$, $p=.03$); low AB, high M, & high FHA greatest reduction
Janes et al. (2010)	21 smokers (women)	Slips 47.7 (8.6) Abstainers 44.23 (12.3)	Modified Stroop (smoking)	Abstinence within 8 week treatment period	8 weeks of NRT & behavioral intervention	**Pre-treatment AB (Wilks's λ = .71) and accuracy effects (Wilks's λ = .60) were significant outcome predictors** $F(1,17) > 6.21$, $p <$ .025)	
Hester et al. (2010**)**	17 methamphetamine dependent individuals	34.3 (sd not reported)	Modified Stroop methamphetamine	Abstinence- 1 mth post discharge	7 days of inpatient treatment with Modafinil or Placebo	**Pre-treatment AB score significantly correlated with days retained in treatmen**t ($r = .60$,$p = .019$)	Self-reported relapse at follow-up ($r = .42$, $p =$ .17)
Powell et al. (2010)	141 smokers	33.2 (12.8)	Modified Stroop (smoking)	Abstinence at 7 days, 30 days, 3 months	Unaided Quit Attempt	**Pre-quit AB predicted relapse** at 7 days**,** Chi square: 6.78., p < .001	AB assessed following overnight abstinence
Garland et al. (2011)	53 Alcohol dependent individuals (inpatient)	Not reported	Spatial Cueing Task (dot probe with distractor)	Relapse at 6 months	Mindfulness vs. Social Support group (control)	**Greater post treatment bias more likely to relapse** *OR* = 1.05 (1.00, 1.11) $p = .03$	AB significantly predicted the rate and timing of relapse, *OR*: 01.04 (95% CI (01.00, 1.08), $p = 0.04$
Carpenter et al. (2012)	25 cocaine dependent individuals	37 (7.1)	Drug Stroop (Cocaine) & Drug-Implicit Relational Assessment Procedure (D-IRAP)	Percentage of sessions attended, amount of voucher/lottery tickets, negative urines	24 week outpatient program-Contingency Management	**AB associated with visits ($r = .51$, $p < .05$), urine** ($r = .49$, $p < .05$), lottery tickets ($r = .48$, $p < .05$) D-IRAP associated with visits ($r = -.47$, $p < .05$), urine ($r = -.52$ $p <.05$)	Measures assessed after 14 days of abstinence

Table 2 Note: AB = Attentional Bias, NRT = nicotine replacement therapy.

Table 3. Attentional Retraining and Smoking

Study	N	No. of trainings	Training Task	Control Condition	Bias Assessment	New Stimuli	Effect on Bias	Effect on Craving	Effect on Smoking	Effect on new pictures/negative bias
Attwood et al., (2008)	27 AR 27 Attend-Smoking	Single session	AR (Attend-Neutral)	Modified VP (Attend-Smoking)	VP task , 1 assessment	Not Included	Main effect of Group: AB lower in AR group	AR reduced cued-craving for males but not for females	No effect on smoking topography	N/A
Field et al., (2009)	24 AR 24 Attend-Smoking 24 Control	Single session	AR (Attend-Neutral)	Modified VP (Attend Smoking, and 50/50)	VP Task, 1 assessment; Stroop, 1 assessment	Included	AB reduced in AR group after training session	No effect on QSU-brief or Urge to smoke scale	No effect on motivation to smoke within 30 minutes or smoking diary	No effect on new pictures
McHugh et al., (2010)	25 AR 26 Control	Single Session	AR (Attend-Neutral)	Modified VP (50/50)	VP task, 1 assessment	Not included	No effect	No effect on QSU-brief	Not assessed	N/A
Kerst et al., (2014)	30 AR 30 Control	~15 over one week	AR (Attend-Neutral)	Modified VP task (50/50)	VP task, ~5 assessments	Not included	AR reduced bias over time	AR reduced craving following smoking cues	No effect on smoking diary or CO	N/A
Lopes et al., (2014)	22 AR 3* 22 AR 1* 22 Control*	3 trainings over two weeks	AR (Attend-Neutral)	Modified VP task with all neutral pictures and VP task 50/50 (new)	VP task , 5 assessments	Included	AR reduced bias 24 hr and 1 mth after training (AR 3 vs. Control)	No effect on single item measure of craving	No effect on smoking diary or CO	Effect on new pictures & avoid 3 group negative bias at $_{6}$ mth
Robinson (2015)	31 AR 33 Control	~30 trainings over two weeks	AR (Attend-Neutral)	Modified VP task (50/50)	VP task , ~10 assessment Stroop, 3 assessments	Included	Main effect of Group: averaged across sessions or days, AB lower in AR group	No effect on cued craving, non-cued craving, or QSU-Brief scores	AR reduced cigarettes smoked on PDA over time. No effect on CO or Cotinine, or smoking diary	Generalized to new pictures & No effect on Self-reported bias/ads

Table 3 Note: AB = Attentional Bias, ABM = Attentional Bias Modification, AR = Attention Retraining, CO = Carbon Monoxide, QSU = Questionnaire for Smoking Urges, VP = Visual Probe; *AR 3 = 3 sessions of AR, AR 1 = 1 session of AR and 2 sessions of placebo (all neutral pictures), AR 0 = 2 placebo session and 1 session of standard VP. For Lopes et al. (2014) participants were assessed at baseline. They were assessed again 24 h, 1, 6 and 12 months after the last ABM session; Participants completed 3 trainings within the first 2 weeks of enrollment to a smoking cessation program, with a 24hr interval between sessions. 50/50 control condition - dots replace smoking and neutral pictures at equal frequency.

Table 4. Summary of meta-analyses on AR

Study	Condition(s)	# of Studies	Participants	Effect Size: Cognitive Bias	Effect Size: Symptoms
Hakamata et al., 2010	Anxiety	12	476	Attentional Bias: large effect, *d* = 1.16	Medium effect, *d* = 0.61
Hallion et al., 2011	Anxiety Depression	45	2,591	Cognitive Bias: large *g* = 0.49 Attentional Bias (*k*=20): medium effect, *g* = 0.29 Interpretation (*k*=20): medium effect, *g* = 0.81	Overall: *g* = 0.13 Overall +stressor: *g* = 0.23 Anxiety: *g* = 0.13 Anxiety +Stressor: *g* = 0.28 Depression + Stressor : *g* = 0.06, ns Depression *g* = 0.06, ns
Beard et al., 2012[2,3,4]	Anxiety Depression, Substance use Self-esteem Eating Pain	Anxiety : 22, Depression: 3, Substance use: 7, Self-esteem: 2, Pain: 1, Eating: 2	2,135	Attentional Bias: Overall: Attend neutral vs attend control : *g* = 0.80 Attend positive vs. attend control : *g* = 0.24 Attend neutral vs. attend-disorder relevant: *g* = 1.19 Threat: Attend neutral vs attend control : *g* = 0.96 Attend neutral vs. attend-disorder relevant: *g* = 1.06 Appetitive : Attend neutral vs attend control : *g* = 0.39 Attend neutral vs. attend-disorder relevant: *g* = 1.41	Post-training: Attend neutral vs control: *g* = 0.01, ns Attend positive vs. control : *g* = 0.09, ns Attend neutral vs. attend-disorder relevant: *g* = 0.03, ns Post-Challenge: Attend neutral vs control: *g* = 0.22, ns Attend positive vs. control : *g* = 0.60, ns Attend neutral vs. attend-disorder relevant: *g* = 0.40 Post-treatment: Attend neutral vs control: *g* = 0.41 Attend positive vs. control : *g* = 0.09
Mogoase et al., 2014[5]	Anxiety Depression Pain Substance Abuse Distress in Healthy	Anxiety: 22 Depression: 7 Pain :1 Substance Abuse: 5, Distress in Healthy: 4	2,268	Attentional Bias: Overall *g* = 0.31 Anxiety: *g* = 0.33 Depression: *g* = 0.22,ns Pain: *g* = 0.20, ns Substance Abuse *g* = 0.34, ns Distress in Healthy : *g* = 0.38	Overall: *g* = 0.160 Overall +stressor: *g* = 0.38 Anxiety: *g* = 0.26 Anxiety +stressor *g* = 0.38 Depression: *g* = -0.11, ns Depression +stressor: *g* = n/a Pain: *g* = -0.15, ns Pain +stressor: *g* = n/a Substance Abuse: .003, ns Substance Abuse +stressor: .06,ns Distress in Healthy: 0 .21 Distress in Healthy +stressor: .51

Table 4 Note. AR = Attention retraining, *g* = Hedge's *g* (a measure of effect size), *k*=number of studies. All effect sizes are significant unless otherwise stated; Conditions refer to the clinical disorder of interest, although many studies included healthy or subclinical samples; Effect size calculations based on: Change Scores (Hakamata et al., 2010; Beard et al., 2012), Post-intervention (Hallion & Ruscio, 2011), and Post-intervention controlling for baseline AB (Mogoase et al., 2014). 1.) Hallion and Ruscio included studies that modified interpretation bias, the attention retraining on symptoms was not reported. 2) Effect sizes were calculated by comparison condition: neutral (e.g., neutral faces) vs. Control, positive (e.g., smiling faces) vs. control, and neutral vs. disorder-relevant (e.g., angry faces/alcohol pictures); 3) Beard et al., reported the effect of AR on symptoms post-training, post-challenge (e.g., an impromptu speech or taste test), and post-treatment (after multiple sessions of AR). 4) Attend Neutral vs. Control: participants in attend neutral group dots always replace neutral stimuli (as opposed to threat/appetitive), control training involved the dot placing neutral or threat/appetitive stimuli at equal frequency; Attend positive vs. control: participants in attend positive group dots always replace positive stimuli (as opposed to threat/appetitive), control same as described above; Attend Neutral vs. Attend Disorder-relevant: participants in attend neutral group dots always replace neutral stimuli (as opposed to threat/appetitive), attend disorder dot always replaces neutral or threat/appetitive stimuli. 5.) Mogoase et al. (2014) excluded studies/condition that trained participants toward disorder-relevant stimuli.

Table 5. Summary of Study Procedures

Study Day →	**Scr.**	**Visit 1 0**	**1-6**	**Visit 2 7**	**7-13**	**Visit 3 14**
Modality/Location	Phone	USUHS	Field	USUHS	Field	USUHS
Inclusion/Exclusion	X	X				
Informed Consent		X				
Randomization		X				
SMARTPHONE INTERVENTION						
3 AR or Control Trainings per day		X	X	X	X	X
QUESTIONNAIRE ASSESSMENTS						
Demographics		X				
Smoking History		X				
WISDM		X				
FTND		X				
QSU		X				
SABQ		X		X		X
Post-treatment Questions						X
SMOKING ASSESSMENTS						
Breath Sample for CO		X		X		X
Saliva Sample for Cotinine		X				X
Smoking Diary (Cigs/day)		X	X	X	X	X
LAB COGNITIVE ASSESSMENTS						
Visual Probe (Old & New Pictures)		X		X		X
Smoking Stroop		X		X		X
Mobile Eye Assessment				X		X
SMARTPHONE ASSESSMENTS						
Visual Probe (Old & New Pictures)		X	X	X	X	X
Self-reported attentional bias		X	X	X	X	X
Self-reported exposure to cues		X	X	X	X	X
Cued Craving		X	X	X	X	X
Non-Cued Craving		X	X	X	X	X
Smoking since last assessment		X	X	X	X	X

Table 5 Note: AR = Attentional retraining; CON = Control trainings; CO = carbon monoxide; FTND = Fagerström Test for Nicotine Dependence ; QSU = Brief Questionnaire for Smoking Urges ; SABQ = Subjective Attentional Bias Questionnaire; WISDM = Wisconsin Inventory of Smoking Dependence Motives

Table 6. Time of AR and Control Intervention

Day	**0**	**0**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**7**	**7**	**8**	**9**	**10**	**11**	**12**	**13**	**14**
Visit	**Visit 1 `**									**Visit 2**								**Visit 3**
AR	VP-Lab		AR1	AR1	AR1	AR1	AR1	AR1	AR1	VP-Lab		AR1	AR1	AR1	AR1	AR1	AR1	VP-Lab
		AR2	AR2	AR2	AR2	AR2	AR2	AR2			AR2	AR2	AR2	AR2	AR2	AR2	AR2	
		AR3	AR3	AR3	AR3	AR3	AR3	AR3			AR3	AR3	AR3	AR3	AR3	AR3	AR3	
		VP	VP	VP	VP	VP	VP	VP			VP	VP	VP	VP	VP	VP	VP	
CON	VP-Lab		C1	C1	C1	C1	C1	C1	C1	VP-Lab		C1	C1	C1	C1	C1	C1	VP-Lab
		C2	C2	C2	C2	C2	C2	C2			C2	C2	C2	C2	C2	C2	C2	
		C3	C3	C3	C3	C3	C3	C3			C3	C3	C3	C3	C3	C3	C3	
		VP	VP	VP	VP	VP	VP	VP			VP	VP	VP	VP	VP	VP	VP	

Table 6 Note: A schematic for the presentation of trainings and assessments for an AR and Control participant. Participants were scheduled to complete 3 trainings and 1 assessment per day. The assessment could occur at the end of the day (as shown above) or earlier in the day. AR = Attentional Retraining; CON = Control training; VP = Visual Probe.

Table 7. Participant Characteristics at Baseline

	AR (n = 31)	CON (n = 33)	t/χ^2	*p*
Age	43.38 (13.41)	43.09 (12.35)	-0.09	.92
Sex (%)			0.23	.63
Male	51.61	48.39		
Female	57.58	42.42		
FTND	4.16 (2.35)	4.64 (2.28)	0.82	.41
QSU	2.68 (1.27)	3.18 (1.22)	1.60	.12
SABQ	1.33 (0.79)	1.72 (0.93)	1.77	.08
Cigarettes per day	12.94 (7.27)	13.33 (7.16)	0.22	.82
WISDM	52.30 (14.76)	58.40 (17.61)	1.50	.13
Age when started daily smoking	20.19 (6.40)	20.12 (6.30)	0.00	.96
Lifetime quit attempts (+24 hrs)	2.94 (5.73)	1.51 (1.97)	-1.34	.18

Table 7 Note. Mean (SD) for Participant Demographics. Continuous variables were examined using *t*-tests. Chi Square values are computed for categorical variables. * = p <.05. AR = Attention retraining, CON = Control, *t* = *t*-value, χ^2 = chi=square, FTND = Fagerström, Test for Nicotine Dependence, QSU = Questionnaire for Smoking Urges, SABQ = Subjective Attentional Bias Questionnaire, WISDM = Wisconsin Inventory of Smoking Dependence Motives.

Table 8. Comparison of Completers (n=49) vs. Non-completers (n=14 at Baseline)

	Non-Completers (n = 14)	Completers (n = 49)	t/χ^2	*p*
Age	41.00 (12.96)	44.22 (12.65)	-0.84	.41
Sex (%)			0.77	.38
Male	64.29	51.02		
Female	35.71	48.98		
FTND	4.21 (2.86)	4.47 (2.19)	-0.36	.72
QSU	3.15 (1.58)	2.91 (1.17)	0.65	.52
SABQ	1.60 (1.22)	1.53 (0.78)	0.29	.77
Cigarettes per day	16.07 (9.52)	12.41 (6.26)	1.36[a]	.19
WISDM	52.87 (20.34)	56.31 (15.49)	-0.68	.50
Age when started daily smoking	21.21 (7.36)	19.94 (6.06)	0.66	.51
Lifetime quit attempts (+24 hrs)	1.21 (1.42)	2.54 (4.77)	-1.02	.31

Table 8 Note. Mean (SD) for Participant Demographics. Continuous variables were examined using *t*-tests. Chi Square (χ^2) values are computed for categorical variables, FTND = Fagerström Test for Nicotine Dependence, QSU = Questionnaire for Smoking Urges, SABQ = Subjective Attentional Bias Questionnaire, WISDM = Wisconsin Inventory of Smoking Dependence Motives. Completers = participants who received AR or Control training and who provided data at the visit 3 laboratory session. Non-completers = participants who did not provide data at the visit 3 session. One participant was excluded from these analyses because his condition assignment (AR vs. Control) was not known. [a]*Welch's t*-test used for cigarettes per day because of unequal variance.

Table 9. Summary Statistics of Lab Dependent Variables by Training Group and Lab Visit

		Visit		
		Base	2	3
AR	VP Bias (n=31, 25, 25)	-6.35 (34.76)	0.09 (26.27)	-15.13 (32.06)
	Old Pictures		5.24 (32.26)	-16.88 (38.38)
	New Pictures		-4.26 (40.45)	-13.38 (40.50)
	Stroop (n=31, 25, 23)	33.56 (116.92)	34.83 (92.04)	38.40 (187.19)
	Reported Attention (n=31,28, 25)	3.75 (1.84)	3.53 (1.99)	3.12 (1.83)
	SABQ (n=31, 29, 25)	1.33 (0.79)	1.19 (0.74)	0.94 (0.76)
	Gazepoints Smoking LZ		11.95 (17.60)	8.53 (11.38)
	Total Gazepoints (n=20,19)		132.25 (16.11)	135.21 (11.84)
	QSU (n=31, 29, 25)	2.69 (1.27)	2.23 (1.25)	2.08 (1.45)
	Cued Craving (n=31, 28,25)	4.48 (2.01)	3.57 (2.08)	3.24 (1.98)
	Craving (n=31,28, 25)	4.00 (2.00)	3.64 (1.99)	3.20 (1.87)
	CO (n=31, 29, 26)	12.16 (5.21)	9.21 (5.98)	9.15 (5.29)
	Cotinine (n=30,29,24)	366.06 (39.36)	415.83 (261.50)	377.03 (259.35)
	Cigarettes Smoked (n=31,28,25)	3.07 (1.60)	3.18 (1.49)	3.28 (1.37)
CON	VP Bias (n=32, 26, 19)	-1.66 (44.87)	15.82 (35.64)	10.33 (29.76)
	Old Pictures		29.62 (56.09)	13.29 (38.13)
	New Pictures		3.02 (34.43)	9.03 (45.08)
	Stroop (n=32, 26,19)	20.86 (120.40)	-5.38 (135.66)	-21.08 (129.62)
	Reported Attention (n=32,27, 23)	4.61 (2.22)	3.93 (2.03)	2.96 (1.72)
	SABQ (n=32,26,21)	1.75 (0.92)	1.51 (0.81)	1.29 (0.82)
	Gazepoints Smoking LZ		6.07 (7.94)	8.92 (9.70)
	Total Gazepoints (n=14,13)		132.00 (24.15)	136.00 (14.32)
	QSU (32,26,21)	3.23 (1.27)	2.60 (1.26)	2.54 (1.21)
	Cued Craving (n=32, 27,23)	4.45 (1.96)	3.52 (2.08)	3.26 (2.20)
	Craving (n=32,27, 23)	4.61 (2.36)	4.11 (1.95)	3.26 (2.16)
	CO (n=32,27,23)	12.19 (4.33)	10.62 (5.52)	10.30 (7.34)
	Cotinine (n=30,26, 22)	388.89 (67.94)	419.1 (237.90)	451.06 (294.60)
	Cigarettes Smoked (n=32,27,23)	2.71 (1.77)	3.56 (1.22)	3.26 (1.49)

Table 9 Note. Mean (SD) for study measures. AR = Attentional Retraining, Base = baseline, CO = expired carbon monoxide, CON = control training, LZ = Smoking Look

Zone, Questionnaire, QSU = Questionnaire for Smoking Urges, SABQ = Subjective Attentional Bias, Stroop = Smoking Stroop Task, VP Bias = Visual Probe attentional bias score (As noted on page 91, subject 30 is excluded from this table).

Table 10. Summary Statistics on EMA Dependent Variables by Training Group and Day

		Day													
		1	2	3	4	5	6	7	8	9	10	11	12	13	14+
AR	VP Bias (ms)	-10.50 (38.43)	9.32 (31.78)	4.77 (52.65)	-16.05 (69.14)	-9.43 (62.16)	-29.13 (121.63)	-9.63 (63.26)	-22.66 (40.86)	-53.35 (215.22)	-15.44 (81.30)	-15.82 (46.14)	-50.23 (131.53)	31.92 (85.05)	20.70 (85.45)
	Reported Attention (1-7)	3.58 (1.95)	3.16 (1.68)	2.73 (1.86)	3.18 (1.92)	3.71 (2.01)	3.12 (2.07)	3.05 (2.03)	3.18 (1.93)	3.05 (2.02)	3.06 (2.17)	2.97 (2.16)	2.75 (1.91)	2.79 (1.92)	3.25 (2.20)
	Advertisements (1-5)	2.30 (1.36)	1.91 (1.33)	1.65 (1.19)	1.73 (1.22)	1.76 (1.29)	1.91 (1.37)	1.86 (1.40)	1.88 (1.39)	1.86 (1.52)	1.98 (1.42)	1.50 (1.40)	1.48 (1.08)	1.82 (1.45)	1.89 (1.47)
	Cued Craving (1-7)	3.60 (1.96)	3.54 (1.94)	3.01 (2.05)	3.10 (2.04)	3.55 (2.01)	3.00 (2.12)	3.24 (2.05)	3.25 (1.95)	3.16 (2.25)	3.08 (2.23)	2.95 (2.21)	2.65 (1.89)	2.45 (1.75)	2.88 (2.18)
	Craving (1-7)	3.69 (1.90)	3.31 (1.75)	2.84 (1.84)	3.18 (1.93)	3.58 (1.95)	2.94 (3.05)	3.20 (2.00	3.21 (2.10)	3.21 (2.10)	3.04 (2.24)	3.13 (2.23)	2.55 (1.87)	2.82 (1.95)	3.32 (2.15)
	Smoking (1-5)	3.42 (1.44)	3.80 (1.44)	3.46 (1.47)	3.25 (1.48)	3.32 (1.53)	3.53 (1.52)	3.38 (1.54)	3.46 (1.54)	3.38 (1.53)	3.17 (1.64)	3.25 (1.53)	3.33 (1.50)	3.20 (1.61)	3.38 (1.39)
	No. Cigs (Diary)	10.85 (6.77)	11.30 (7.06)	11.56 (7.64)	12.48 (8.45)	12.11 (8.59)	11.67 (8.33)	10.19 (8.89)	10.54 (7.28)	10.13 (7.38)	9.96 (7.74)	10.79 (9.15)	10.82(9.29)	10.22 (7.80)	12.44 (10.79)
CON	VP Bias (ms)	-15.19 (45.75)	17.29 (50.60)	35.77 (101.32)	18.93 (39.93)	-1.13 (106.93)	10.10 (82.20)	4.03 (77.72)	40.32 (118.51)	4.14 (27.15)	2.29 (55.99)	-16.61 (58.42)	51.78 (200.65)	15.75 (43.67)	21.44 (208.19)
	Reported Attention (1-7)	3.72 (2.00)	3.53 (1.82)	3.75 (1.83)	3.28 (1.80)	3.40 (1.82)	3.36 (1.92)	3.24 (1.943)	3.45 (2.07)	3.48 (2.01)	2.77 (1.71)	2.97 (1.71)	2.97 (1.85)	3.13 (2.06)	2.99 (1.73)
	Advertisements (1-5)	1.91 (1.13)	1.88 (1.21)	1.65 (1.11)	1.76 (1.32)	1.62 (1.14)	1.59 (1.02)	1.56 (.94)	1.70 (1.07)	1.66 (.99)	1.58 (1.06)	1.70 (1.27)	1.63 (1.09)	1.65 (1.13)	1.74 (1.27)
	Cued Craving (1-7)	3.83 (2.05)	4.22 (2.09)	3.94 (1.86)	3.43 (1.88)	3.34 (1.97)	3.37 (1.99)	3.45 (2.21)	3.49 (2.02)	3.70 (2.17)	2.94 (1.94)	3.26 (2.00)	3.11 (2.10)	3.24 (2.15)	3.13 (1.98)
	Craving (1-7)	3.93 (2.11)	3.95 (2.06)	3.84 (1.86)	3.42 (1.83)	3.44 (1.96)	3.48 (1.97)	3.38 (2.10)	3.76 (2.12)	3.71 (2.16)	2.98 (1.88)	3.06 (1.98)	3.22 (2.04)	3.16 (2.11)	3.14 (1.82)
	Smoking (1-5)	3.28 (1.50)	3.31 (1.51)	3.31 (1.49)	3.33 (1.51)	3.17 (1.44)	3.58 (1.48)	3.29 (1.59)	3.42 (1.34)	3.34 (1.43)	3.48 (1.47)	3.59 (1.49)	3.5 (1.34)	3.53 (1.42)	3.38 (1.44)
	No. Cigs (Diary)	10.74 (5.73)	12.07 (5.90)	10.63 (6.52)	10.00 (6.66)	10.11 (6.20)	10.77 (7.20)	10.07 (6.91)	9.88 (5.78)	11.00 (6.54)	10.58 (6.07)	10.83 (6.88)	10.56 (6.81)	10.27 (6.95)	10.53 (7.17)

Table 10 Note. Mean (SD) for study measures. Data derive from EMA assessments Days 1 - 14 or smoking diaries. CON = control training, No. Cigs (Diary) = Number of cigarettes reported in smoking diary, per day, VP Bias = Visual Probe

attentional bias score. For advertisements 1= No advertisements seen, 2 = 1 advertisements seen therefore 1.91 is approximately 1 advertisement seen.

Table 11. Summary Statistics for New Pictures in the Lab and Field

		Week 1		Week 2	
		EMA	Lab	EMA	Lab
AR	VP Bias (ms)	-5.77 (52.67)	-4.26 (40.45)	-38.83 (196.02)	-13.38 (40.50)
CON	VP Bias (ms)	29.32 (58.08)	3.02 (56.09)	30.54 (41.59)	9.03 (45.08)

Table 11 Note. VP Bias = Visual Probe attentional bias score; Mean (SD) for VP attentional bias measures. Sample sizes for EMA data are n=15 (AR Week1), n=15 (AR Week2), n=17 (CON Week 1), n=13 (CON Week 2). Sample sizes for Lab data are n=23 (AR Week1), n=25 (AR Week2), n=24 (CON Week 1), n=17 (CON Week 2).

Table 12. Results of LMMs for Laboratory data

	n_1	n_2	Group					Group by Visit				
			df	*PE*	*SE*	*F*	*p*	*df*	*PE*	*SE*	*F*	*p*
VP Bias	183	55	1,126	19.24	6.34	9.20	**.003**	1,125	-10.02	12.14	0.68	.41
Stroop	93	55	1,37	-51.39	32.94	2.43	.13	1,36	28.73	49.35	0.34	.56
Reported Attention	103	57	1,45	-0.36	0.37	.99	.33	1,44	0.44	0.62	0.51	.48
SABQ	101	56	1,44	0.07	0.15	0.25	.62	1,43	0.02	0.21	0.01	.93
LZ Gazepoints	66	46	1.19	-2.84	3.21	0.79	.39	1,18	-6.28	6.42	.96	.34
QSU	101	56	1,44	-0.13	0.24	0.28	.60	1,43	-0.04	0.25	0.03	.86
Cued Craving	103	57	1,45	-0.03	0.46	0.00	.95	1,44	0.03	0.55	0.00	.95
Craving	103	57	1,45	-0.01	0.40	0.00	.99	1,44	0.35	0.61	0.33	.57
Smoking	103	57	1,45	0.25	0.32	0.60	.44	1,44	0.39	0.41	0.90	.35
CO	105	57	1,47	0.54	1.25	0.19	.67	1,46	-0.02	1.50	0.00	.99
Cotinine	101	57	1,43	11.39	46.87	0.06	.81	1,42	-51.25	50.58	0.32	.57

Table 12 Note. n_1 = no. of assessments; n_2 = number of subjects who completed at least one assessment at visit 2 or visit 3. Analyses included all subjects who participated in at least one visit post-treatment. The columns labeled Group show the results for the main effect of Group. The comparison category is AR (Positive parameter estimates indicate higher values for Control vs. AR). The column labeled Group by Visit shows the results for the Group by Visit interaction term. Visit is a categorical variable with two levels (visit 2 vs. visit 3). All models include main effects for Group and Visit. The LMM for bias also includes Picture Type (old vs. new) as an additional within-subject factor. In addition, the baseline (pre-intervention) measure for each dependent variable was included as a covariate. (No covariate was included for LZ Gazepoints because the eye tracking assessment was not administered at baseline). Key: CO = expired carbon monoxide, LZ = Smoking Look Zone, Questionnaire, QSU = Questionnaire for Smoking Urges, SABQ = Subjective Attentional Bias Questionnaire, VP Bias =

Visual Probe attentional bias score, PE = (unstandardized) parameter estimate; SE = standard error; F = F value from mixed model.

Table 13. Results of LMMs for EMA data

	n_1	n_2	Group					Group by Day				
			df	*PE*	*SE*	*F*	*p*	*df*	*PE*	*SE*	*F*	*p*
VP Bias (ms)	481	53	1, 374	24.06	9.68	6.18	**.01**	1, 374	-0.34	2.63	0.02	.90
Reported Attention	2249	56	1, 2081	0.23	0.23	0.95	.33	1, 2081	-0.02	0.02	1.12	.29
Advertisements	2241	56	1, 2075	-0.05	0.12	0.20	.65	1, 2075	-0.00	0.12	0.00	.96
Cued Craving	2276	56	1, 2108	0.45	0.28	2.50	.11	1, 2108	-0.02	0.03	0.44	.51
Craving	2276	56	1, 2108	0.39	0.28	2.00	.16	1, 2108	-0.03	0.03	1.20	.27
Smoking (PDA)	2225	56	1, 2057	-0.02	0.24	0.01	.93	1, 2057	0.04	0.02	3.91	**.04**
No. Cigs (Diary)	738	54	1, 683	-1.25	1.07	1.38	.24	1, 682	0.01	0.05	0.05	.82

Table 13 Note. n_1 = no. of assessments or days (No. Cigs); n_2 = number of subjects. Analyses included all subjects who provided EMA data. The columns labeled Group show the results for the main effect of Group. The comparison category is AR (Positive parameter estimates indicate higher values for Control vs. AR).The columns labeled Group by Day show the results for the Group by Day interaction term. In addition, the baseline (pre-intervention) measure for each dependent variable was included as a covariate. The data for analysis of bias use all assessments with no more than one reported interruption (see text). Key: No. Cigs (Diary) = Number of cigarettes reported in smoking diary, VP Bias = Visual Probe attentional bias score, PE = (unstandardized) parameter estimate; SE = standard error; F = F value from mixed model.

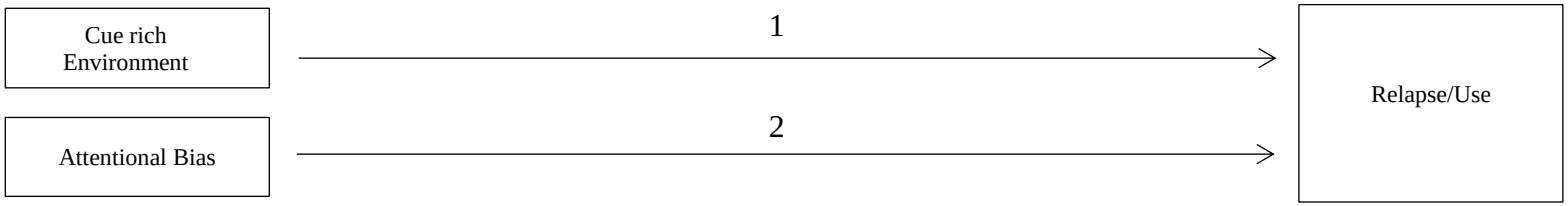


Figure 1: Preliminary Model

Note. Both a cue rich environment ("route 1") and attentional bias ("route 2") may provoke relapse/use in African American smokers.

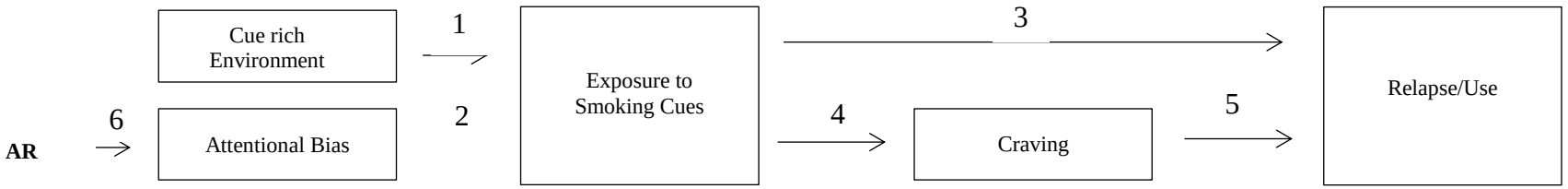


Figure 2. Conceptual Model

Note. AR = Attentional retraining. Exposure to smoking cues in the real world can be increased by a cue-rich environment and/or greater attentional bias. It was proposed that exposure to smoking cues in smokers' natural environments could be reduced by reducing attentional bias to smoking cues. Reduced exposure should lead to reduced craving and reduced use. Evidence for pathways 1 to 6 is provided in the text. Note that the term "pathway" is used in Figure 2 to differentiate them from the "routes" illustrated in Figure 1.

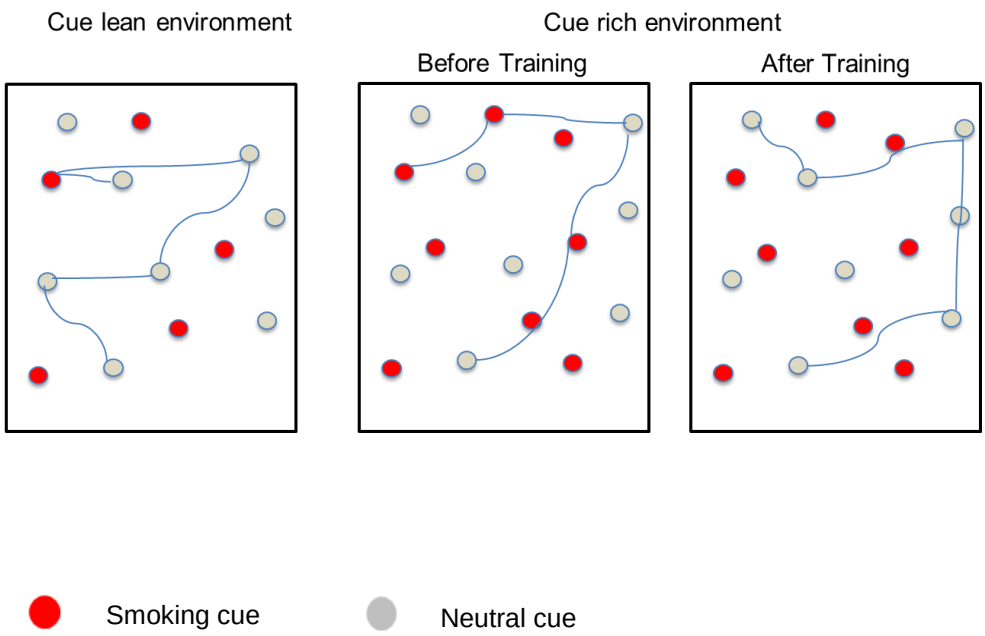


Figure 3. Proposed effect of AR

Note. AR = Attention retraining. The first panel depicts a cue lean environment in which there are few smoking cues. The second panel depicts a cue rich environment in which there are many smoking cues. This environment is pertinent to African American smokers. The third panel depicts a cue rich environment after a smoker has received AR. Smokers in the third panel attend less to smoking cues which makes their environment similar to a cue lean environment. AR in African American smokers should reduce attentional bias to smoking cues, and therefore exposure to smoking cues in their natural environment. The environment has not changed, but their perception of the environment has changed.

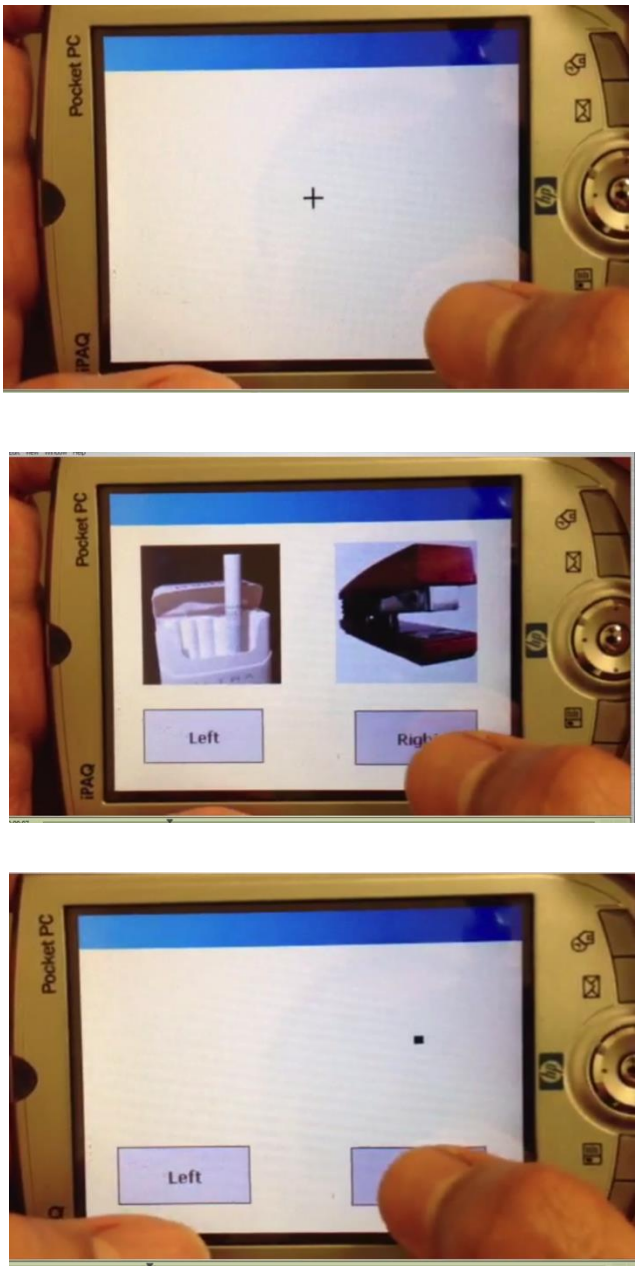
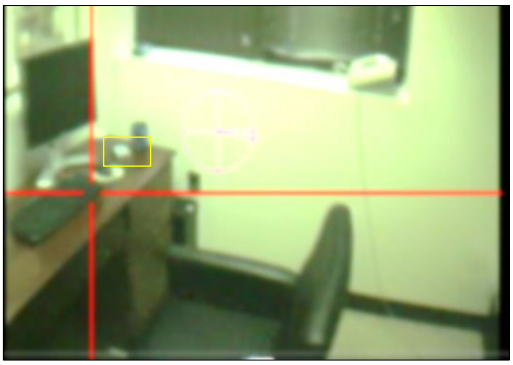


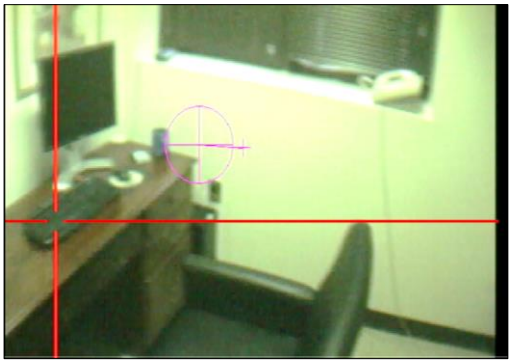
Figure 4. Diagram of events in a VP trial

Note. VP = Visual Probe. 1) A fixation cross is presented for 500ms, 2) The picture pair (neutral and smoking) are displayed (500 ms); and 3) The probe to which the participant must respond (left or right) is presented. For the standard VP task (and Control Task) the probe is equally likely to replace the smoking or neutral picture. For the AR task the probe replaces neutral stimuli on all trials. On the standard VP task, attentional bias is

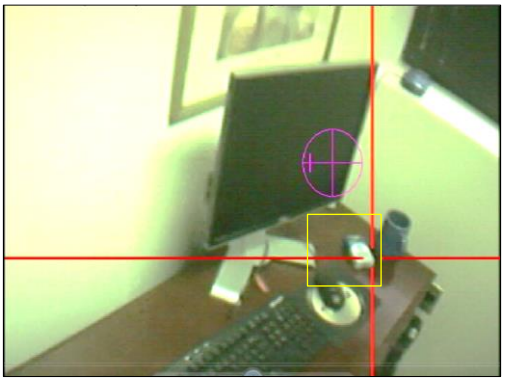
computed as the difference in RTs on trials where the probe replaces the smoking picture vs. trials where the probe replaces the neutral picture. This formula yields an attentional bias score in which high (positive) values correspond to an attentional bias toward smoking stimuli and low (negative) values correspond to an attentional bias away from smoking stimuli and toward neutral stimuli ("avoidance").



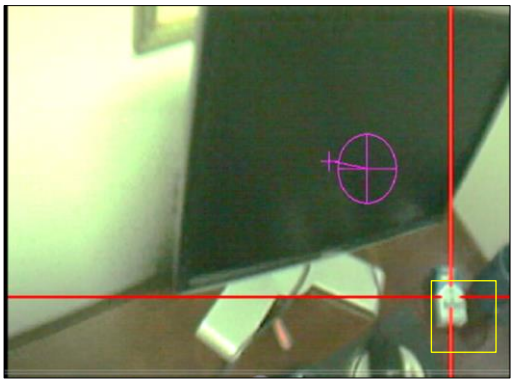
Screenshot 1.
Time: 1:30



Screenshot 2.
Time: 2:03

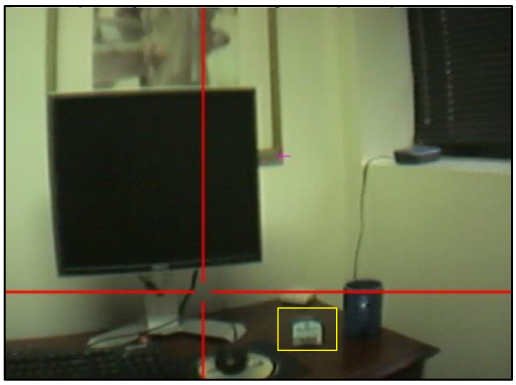


Screenshot 3.
Time: 3:83



Screenshot 4.
Time: 4:22

5



Screenshot 5.
Time: 5:00

Figure 5. Mobile Eye Tracker Screen Shots

Note: Sample screenshots of five video frames over 5.00 seconds of mobile eye task. Gaze tracking (where the participant is fixating) is indicated by the red circle and crosshairs. Smoking Lookzone (i.e., the smoking stimuli) is in yellow. The elapsed time is presented under each video. The purple circle represents the reflection of the pupil, information from the gaze is used to determine where the fixations cross should appear.

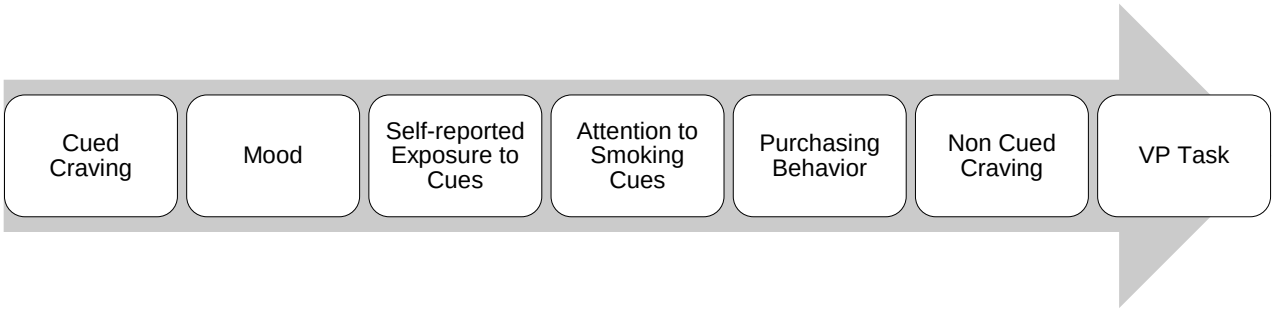


Figure 6. Schematic Depiction of a PDA field assessment

Note. PDA = Personal Digital Assistant, VP = Visual Probe, AR = Attention retraining. During each Personal Digital Field Assessment field assessment, the participant answered the questions in the order above and completed the VP task. The VP task was either AR, a Control task, or the Assessment.

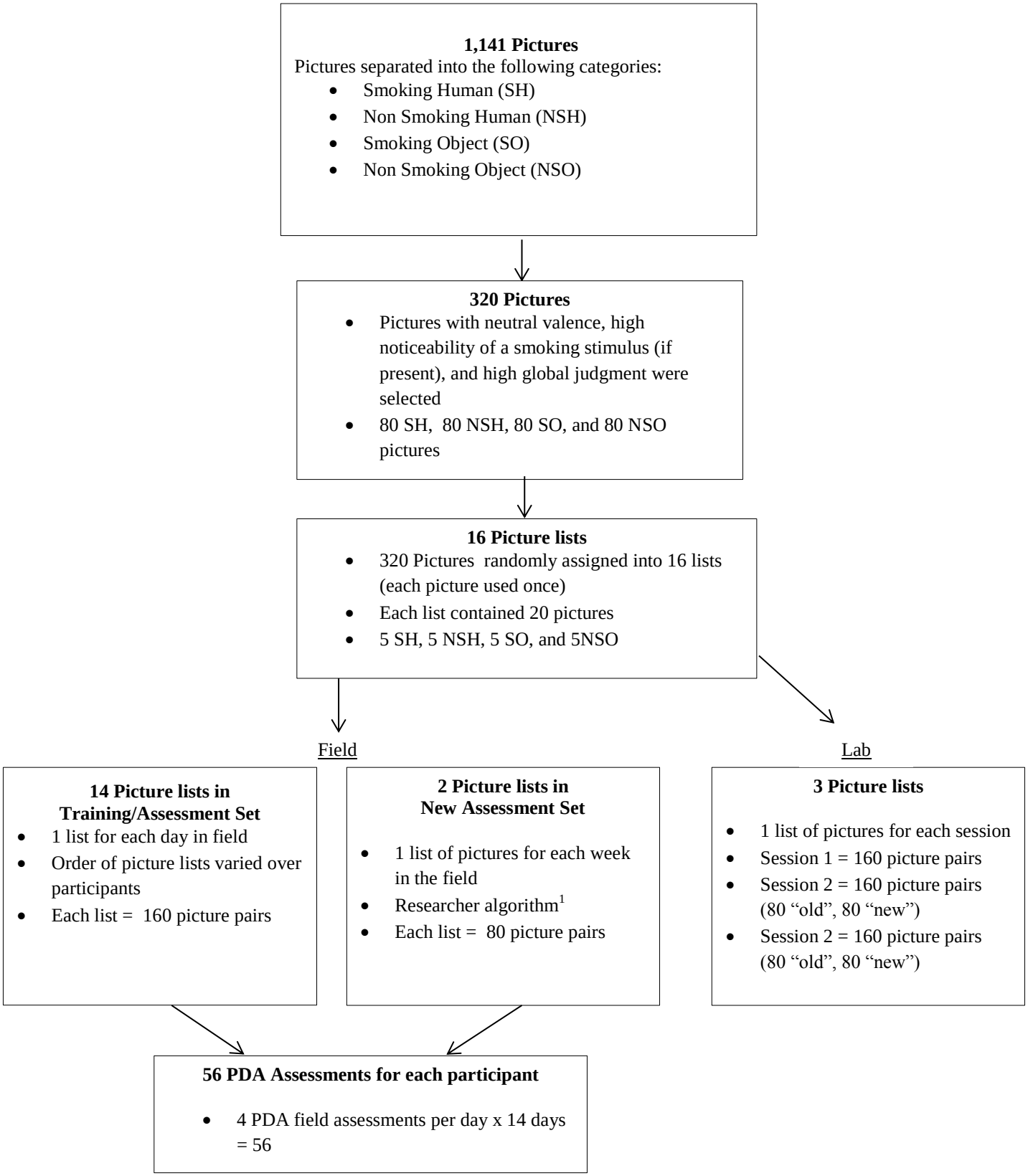

1,141 Pictures
Pictures separated into the following categories:
Smoking Human (SH)
Non Smoking Human (NSH)
Smoking Object (SO)
Non Smoking Object (NSO)
320 Pictures
Pictures with neutral valence, high noticeability of a smoking stimulus (if present), and high global judgment were selected
80 SH, 80 NSH, 80 SO, and 80 NSO pictures
16 Picture lists
320 Pictures randomly assigned into 16 lists (each picture used once)
Each list contained 20 pictures
5 SH, 5 NSH, 5 SO, and 5NSO
Field
Lab
14 Picture lists in Training/Assessment Set
1 list for each day in field
Order of picture lists varied over participants
Each list = 160 picture pairs
2 Picture lists in New Assessment Set
1 list of pictures for each week in the field
Researcher algorithm[1]
Each list = 80 picture pairs
3 Picture lists
1 list of pictures for each session
Session 1 = 160 picture pairs
Session 2 = 160 picture pairs (80 "old", 80 "new")
Session 2 = 160 picture pairs (80 "old", 80 "new")
56 PDA Assessments for each participant
4 PDA field assessments per day x 14 days = 56

Figure 7. Flowchart for creation of pictures, training, and control assessment

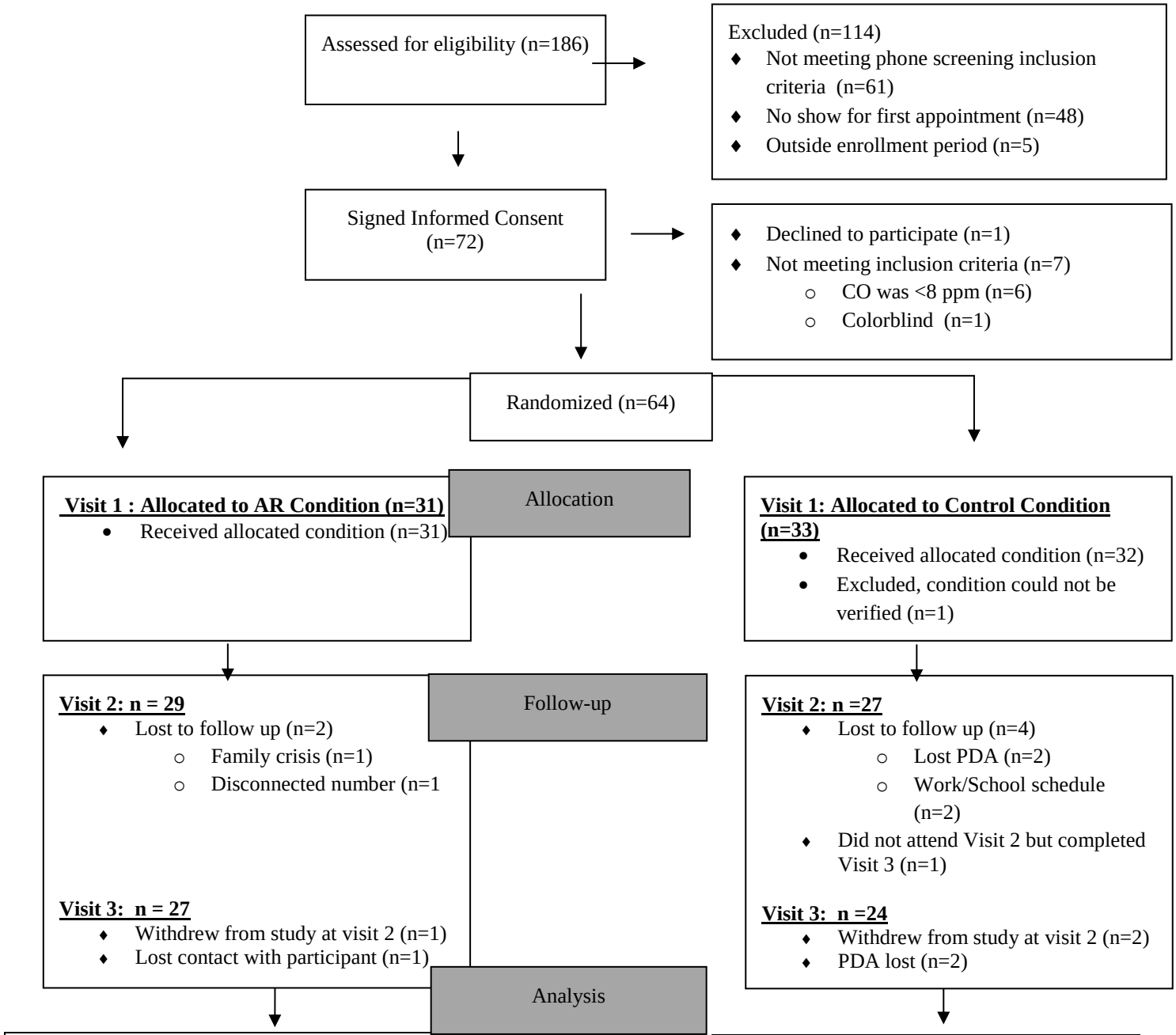
Note: Picture lists were randomly selected from the 14 Pictures Training/Assessment set.

[1]Participants were scheduled to see new pictures according to algorithm in Figure 8.

Participant Randomization File (AR or Control)	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**	**11**	**12**	**13**	**14**	**15**	**16**	**17**	**18**	**19**	**20**	**21**	**22**	**23**	**24**	**25**	**26**	**27**	**28**	**29**	**30**
Wk1Day	1	2	3	4	5	6	7	1	2	3	4	5	6	7	1	2	3	4	5	6	7	1	2	3	4	5	6	7	3	5
Wk2Day	8	9	10	11	12	13	14	8	9	10	11	12	13	14	8	9	10	11	12	13	14	8	9	10	11	12	13	14	10	12

Figure 8. Algorithm for new picture assignment

Note: Participants were assigned to receive new pictures during the 4th PDA field assessment on one day of each week. For example, the participants assigned to file "5" received the new picture assessment on day 5 in Week 1 and on day 12 in Week 2. Participants were randomly assigned to files.



Variable↓	Visit 1 (n)	Visit 2 (n)	Visit 3 (n)
CO	31	29	26[d1]
Cotinine	30[c1]	29	24[b2, d1]
QDS	31	28[b1]	25[c1,d1]
VP	27[a1,b3]	25[a1,b2,e1]	25 [b1,d1]
Lab Self-report	29[a1,b1]	28[a1]	25[a1,d1]
Stroop	30[a1]	25[a3,e1]	23[a2,d1,e1]

	Wk 1 (n)	Wk 2 (n)
Diary	28[d1]	26[d1]
EMA	28[a1,c2]	24[a1]
Mobile eye	20[a8,c1]	19[a6,c2]

Variable↓	Visit 1 (n)	Visit 2 (n)	Visit 3 (n)
CO	32[b1]	27	23[d1]
Cotinine	30[a2,b1]	26[b1]	22[b1,d1]
QDS	32[b1]	25[b2]	22[b1,d1]
VP	25[a2,b1,e5]	26[e1]	19[a1,b1,d1,e2]
Lab Self-report	31[b2]	27	23[d1]
Stroop	29[a1,b1,e2]	26[a1]	19[a1,b2,d1,e1]

	Wk 1 (n)	Wk 2 (n)
Diary	27	23[d1]
EMA	28	26
Mobile Eye	14[a7,c6]	13[a11]

Figure 9. CONSORT Flow Diagram

Coding system for missing data: a = technical error, b = researcher error, c = participant error, d = participant refused to complete, e = statistical factor (extreme scores, interruptions, errors). Numbers reflect number of cases (e.g., b2 = data from 2 participants were lost due to participant error). Field week 1= 1-7; Field week 2 = 8+

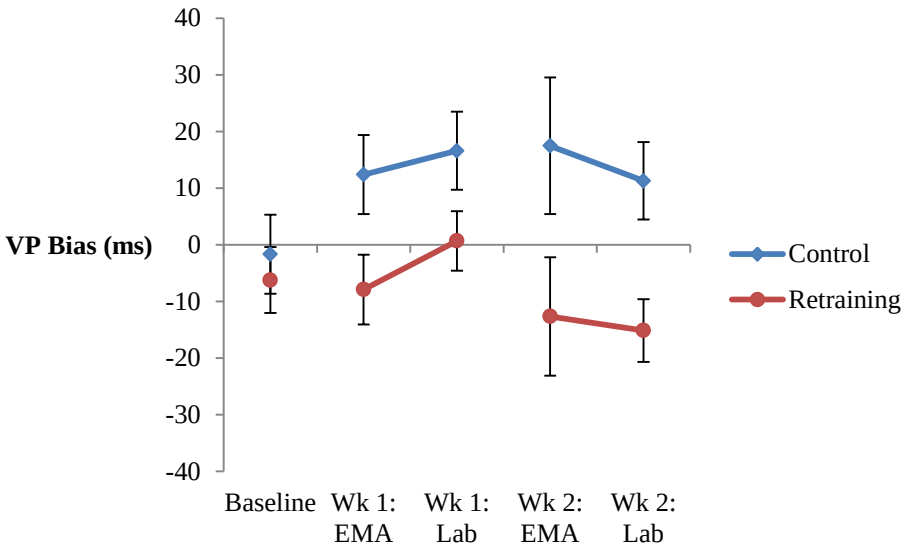


Figure 10. VP task assessed during EMA and lab sessions.

Note: VP Bias = Visual Probe attentional bias score, EMA = Ecological Momentary Assesment, Wk = Week. Data for EMA are aggregated over all assessments during Wk 1 or Wk 2. Data are also aggregated over old and new pictures (both EMA and lab). Wk1 Lab = Visit 2 lab session. Wk 2 Lab = Visit 3 lab session (final session). Error bars are ±1 SE.

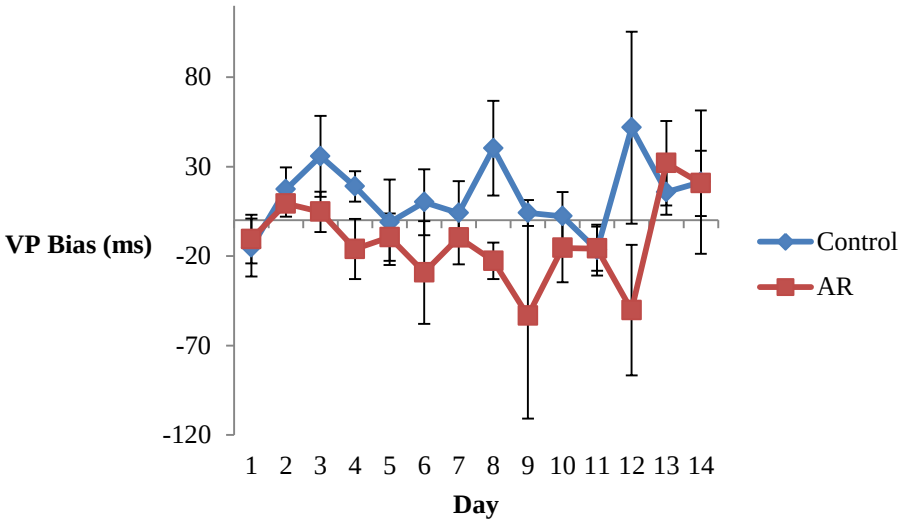


Figure 11. Effect of Group and Day on attentional bias on PDA field assessments

Note: Error bars are ±1 SE. VP Bias = Visual Probe attentional bias score, AR = Attention retraining.

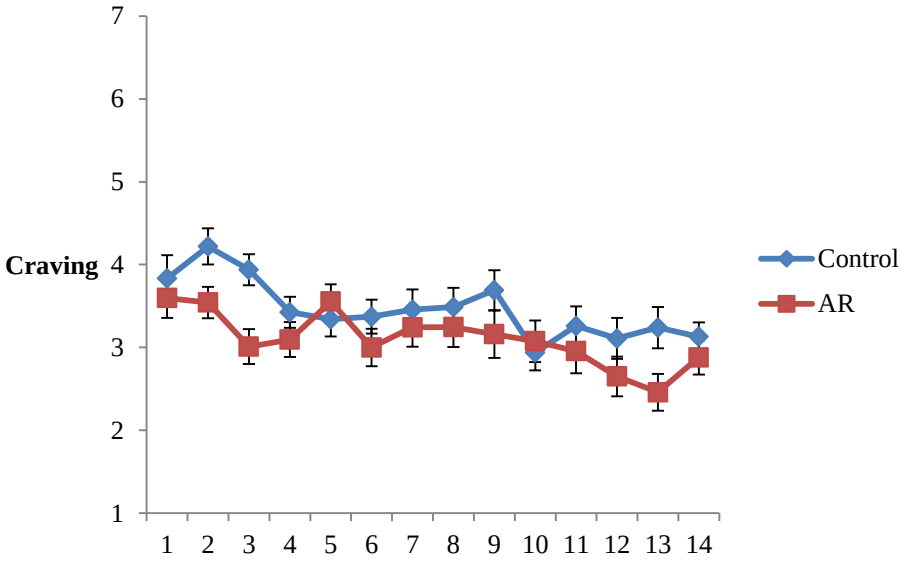


Figure 12. Effect of Group and Day on cued craving (1-7 scale) on PDA field assessments

Note: Error bars are ±1 SE. AR = Attention retraining

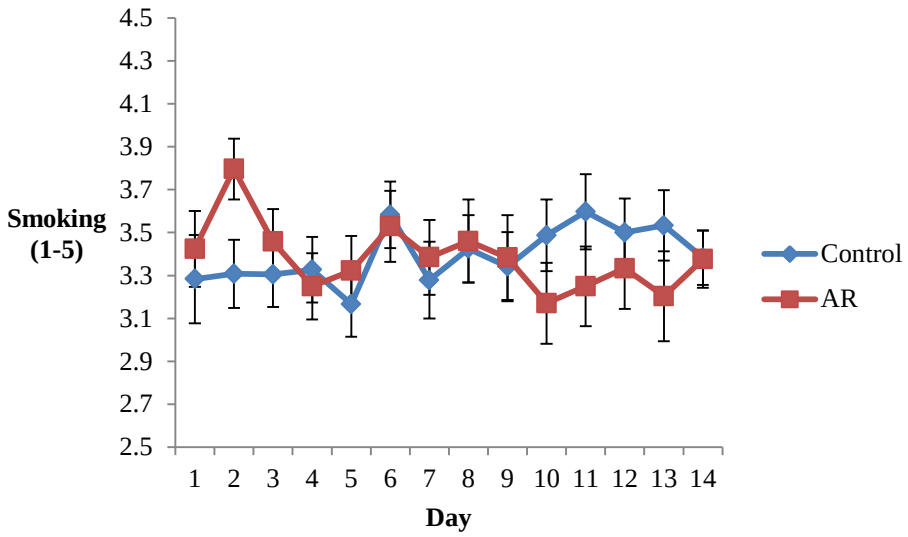


Figure 13. Effect of Group and Day on reported smoking (1-5 scale) on PDA field assessments. AR = Attention retraining

Appendix A. Measures

Demographics Questionnaire

How old are you?

DEM_Q1 How old are you?

0 - 96 = range

What is your gender?

DEM_Q2 What is your gender?

1 = Male
2 = Female

What is your present marital status? (Choose one)

DEM_Q3 What is your marital status?

1 = Single
2 = Married
3 = Divorced
4 = Widowed
5 = Living with a significant other
6 = Separated
8 = Refuse to answer

How many years of education have you completed? (Choose one)

DEM_Q4 How many years of education have you completed? 2

1 = 1 (Elementary School)
2 = 2 (Elementary School)
3 = 3 (Elementary School)
4 = 4 (Elementary School)
5 = 5 (Elementary School)
6 = 6 (Middle School)
7 = 7 (Middle School)
8 = 8 (Middle School)
9 = 9 (High School)
10 = 10 (High School)
11 = 11 (High School)
12 = 12 (High School)
13 = 13 (Some College)
14 = 14 (Vocational or Community College degree)
16 = 16 (Four Year College Degree)
17 = 17 (Some Postgraduate Work)
18 = 18 (Postgraduate Degree: Master Degree)
20 = 20 (OPostgraduate Degree: M.D., Ph.D., DDS, Dr.P.H., etc.)
98 = Refuse to Answer

Are you of Hispanic/Latino origin?

DEM_Q5 Are you of Hispanic/Latino origin?

0 = No
1 = Yes
8 = Refuse to Answer

What category best describes your race? (Choose one)

DEM_Q6 What category best describes your race?

1 = Anglo American/Euro American/White
2 = African American/ Black
3 = Asian American
4 = Native of Hawaii or other Pacific Islander
5 = Native American or Alaska Native
6 = Mixed Race
7 = Other
8 = Refuse to Answer

Please specify your race_ __ __ __ __ __ __

DEM_Q7 Please specify your race

Do you receive Medicare, Medicaid, or Medical Assistance currently?

DEM_Q8 Do you receive Medicare, Medicaid, or Medical Assistance currently?

0 = No
1 = Yes
7 = Don't Know
8 = Refuse to Answer

Do you have private insurance or group insurance?

DEM_Q9 Do you have private insurance or group insurance?

0 = No
1 = Yes
7 = Don't Know
8 = Refuse to Answer

What is you total family income per year, before taxes?

DEM_10 What is you total family income per year, before taxes?

1 = Less than $10,000 per year or less than about $833 per month
2 = $10,000 to $19,999 per year or less than about $1250 per month
3 = $20,000 to $29,999 per year or less than about $2083 per month
4 = $30,000 to $39,999 per year or less than about $2916 per month
5 = $40,000 to $49,999 per year or less than about $3750 per month
6 = $50,000 to $59,999 per year or less than about $4583 per month
7 = $60,000 to $69,999 per year or less than about $5416 per month
8 = $70,000 to $79,999 per year or less than about $6250 per month
9 = $80,000 to $89,999 per year or less than about $7083 per month
10 = $90,000 to $99,999 per year or less than about $7916 per month
11 = $100,000 or more per year or more than $8333 per month
98 = Refuse to Answer

Generations in the U.S. Please choose the best response:

DEM_Q11 Generations in the U.S. Please choose the best response:

1 = I'm an immigrant of the US
2 = I was born in the US
3 = One of my parents and I were born in the US (the other parent immigrated)
4 = My parents and I were born in the US
5 = My grandparents, my parents, and I were born in the US
6 = My great-grandparents and ancestors were born in the US
8 = Refuse to Answer

Employment Status. Please choose the best response:

DEM_Q13 Employment Status. Please choose the best response:

1 = Regular full-time (30 or more hours per week)
2 = Regular part-time (less than 30 hours per week)
3 = Unemployed, currently *looking* for work
4 = Unemployed, currently *NOT looking* for work
5 = Homemaker
6 = Student
7 = Retired
8 = Unable to work or disabled
9 = Other
98 = Refuse to Answer

Please specify your employment status.

DEM_Q14 Please specify your employment status.

In the past 30 days, what was the primary source of your income?

DEM_Q15 In the past 30 days, what was the primary source of your income?

1 = A job
2 = Unemployment Benefits
3 = VA/Disability/Social Security Income
4 = Welfare/Food Stamps/Aif to Family with Dependent Children
5 = Alimony or Child Support
6 = Spouse/partner is main source of income
8 = Refuse to Answer

Smoking History Questionnaire

About how old were you when you first started smoking at least 1 cigarette a day?

SH_Q1 About how old were you when you first started smoking at least 1 cigarette a day?

0 - 96 = range

About how old were you when you started smoking regularly <u>everyday</u>?

SH_Q2 About how old were you when you started smoking regularly everyday?

0 - 96 = range

How many cigarettes do you smoke on a <u>normal day</u>?

SH_Q3 How many cigarettes do you smoke on a normal day?

0 - 96 = range

Do you think you are addicted to smoking?

SH_Q4 Do you think you are addicted to smoking?

0 = Definitely not
1 = Probably not
2 = Possibly
3 = Probably
4 = Definitely

Are you seriously thinking of quitting smoking?

SH_Q5 Are you seriously thinking of quitting smoking?

1 = Yes, within the next 30 days
2 = Yes, within the next 6 months
3 = No, not thinking of quitting

Have you used other tobacco products (i.e. cigars, pipes, smokeless tobacco, bidis, cloves)?

SH_Q6 Have you used other tobacco products (i.e. cigars, pipes, smokeless tobacco, bidis, cloves)?

0 = No
1 = Yes

. **Describe:**

SH_Q6B Describe

Have you ever made a serious and deliberate attempt to STOP SMOKING cigarettes completely?

SH_Q7 Have you ever made a serious and deliberate attempt to STOP SMOKING cigarettes completely?

0 = No
1 = Yes

If so, how many times?

SH_Q8 If so, how many times?

0 - 96 = range

In the <u>last year</u>, how many times have you quit smoking for at least 24 hours?

SH_Q9 In the last year, how many times have you quit smoking for at least 24 hours?

0 - 96 = range

How hard was it for you to quit smoking on your most recent attempt?

SH_Q10 How hard was it for you to quit smoking on your most recent attempt?

1 = Easy
2 = Slighlty Difficult
3 = Difficult
4 = Very Difficult
9 = Not Applicable

Cravings for cigarettes

SH_Q11 Cravings for cigarettes

1 = Not at all
2 = Mild
3 = Moderate
4 = Severe
5 = Very severe

Irritability

SH_Q12 Irritability

1 = Not at all
2 = Mild
3 = Moderate
4 = Severe
5 = Very severe

Nervousness

SH_Q13 Nervousness

1 = Not at all
2 = Mild
3 = Moderate
4 = Severe
5 = Very severe

Difficulty concentrating

SH_Q14 Difficulty concentrating

1 = Not at all
2 = Mild
3 = Moderate
4 = Severe
5 = Very Severe

Physical symptoms

SH_Q15 Physical symptoms

1 = Not at all
2 = Mild
3 = Moderate
4 = Severe
5 = Very severe

Difficulty sleeping

SH_Q16 Difficulty sleeping

1 = Not at all
2 = Mild
3 = Moderate
4 = Severe
5 = Very severe

Wisconsin Inventory of Smoking Dependence Motives-68 (Piper et al., 2004)

Below are a series of statements about cigarette smoking. Please rate your level of agreement for each using the following scale:

1	2	3	4	5	6	7
Not True of Me At All						Extremely True of Me

1.	I enjoy the taste of cigarettes most of the time.	1	2	3	4	5	6	7
2.	Smoking keeps me from gaining weight.	1	2	3	4	5	6	7
3.	Smoking makes a good mood better.	1	2	3	4	5	6	7
4.	If I always smoke in a certain place it is hard to be there and not smoke.	1	2	3	4	5	6	7
5.	I often smoke without thinking about it.	1	2	3	4	5	6	7
6.	Cigarettes control me.	1	2	3	4	5	6	7
7.	Smoking a cigarette improves my mood.	1	2	3	4	5	6	7
8.	Smoking makes me feel content.	1	2	3	4	5	6	7
9.	I usually want to smoke right after I wake up.	1	2	3	4	5	6	7
10.	Very few things give me pleasure each day like cigarettes.	1	2	3	4	5	6	7
11.	It's hard to ignore an urge to smoke.	1	2	3	4	5	6	7
12.	The flavor of a cigarette is pleasing.	1	2	3	4	5	6	7
13.	I smoke when I really need to concentrate.	1	2	3	4	5	6	7
14.	I can only go a couple hours between cigarettes.	1	2	3	4	5	6	7
15.	I frequently smoke to keep my mind focussed.	1	2	3	4	5	6	7
16.	I rely upon smoking to control my hunger and eating.	1	2	3	4	5	6	7
17.	My life is full of reminders to smoke.	1	2	3	4	5	6	7
18.	Smoking helps me feel better in seconds.	1	2	3	4	5	6	7
19.	I smoke without deciding to.	1	2	3	4	5	6	7
20.	Cigarettes keep me company, like a close friend.	1	2	3	4	5	6	7
21.	Few things would be able to replace smoking in my life.	1	2	3	4	5	6	7
22.	I'm around smokers much of the time.	1	2	3	4	5	6	7
23.	There are particular sights and smells that trigger strong urges to smoke.	1	2	3	4	5	6	7
24.	Smoking helps me stay focussed.	1	2	3	4	5	6	7
25.	Smoking helps me deal with stress.	1	2	3	4	5	6	7
26.	I frequently light cigarettes without thinking about it.	1	2	3	4	5	6	7
27.	Most of my daily cigarettes taste good.	1	2	3	4	5	6	7
28.	Sometimes I feel like cigarettes rule my life.	1	2	3	4	5	6	7
29.	I frequently crave cigarettes.	1	2	3	4	5	6	7
30.	Most of the people I spend time with are smokers.	1	2	3	4	5	6	7
31.	Weight control is a major reason that I smoke.	1	2	3	4	5	6	7
32.	I usually feel much better after a cigarette.	1	2	3	4	5	6	7
33.	Some of the cigarettes I smoke taste great.	1	2	3	4	5	6	7
34.	I'm really hooked on cigarettes.	1	2	3	4	5	6	7
35.	Smoking is the fastest way to reward myself.	1	2	3	4	5	6	7
36.	Sometimes I feel like cigarettes are my best friends.	1	2	3	4	5	6	7

37. My urges to smoke keep getting stronger if I don't smoke.	1	2	3	4	5	6	7
38. I would continue smoking, even if it meant I could spend less time on my hobbies and other interests.	1	2	3	4	5	6	7
39. My concentration is improved after smoking a cigarette.	1	2	3	4	5	6	7
40. Seeing someone smoke makes me really want a cigarette.	1	2	3	4	5	6	7
41. I find myself reaching for cigarettes without thinking about it.	1	2	3	4	5	6	7
42. I crave cigarettes at certain times of day.	1	2	3	4	5	6	7
43. I would feel alone without my cigarettes.	1	2	3	4	5	6	7
44. A lot of my friends or family smoke.	1	2	3	4	5	6	7
45. Smoking brings me a lot of pleasure.	1	2	3	4	5	6	7
46. Cigarettes are about the only things that can give me a lift when I need it.	1	2	3	4	5	6	7
47. Other smokers would consider me a heavy smoker.	1	2	3	4	5	6	7
48. I feel a strong bond with my cigarettes.	1	2	3	4	5	6	7
49. It would take a pretty serious medical problem to make me quit smoking.	1	2	3	4	5	6	7
50. When I haven't been able to smoke for a few hours, the craving gets intolerable.	1	2	3	4	5	6	7
51. When I do certain things I know I'm going to smoke.	1	2	3	4	5	6	7
52. Most of my friends and acquaintances smoke.	1	2	3	4	5	6	7
53. I love the feel of inhaling the smoke into my mouth.	1	2	3	4	5	6	7
54. I smoke within the first 30 minutes of awakening in the morning.	1	2	3	4	5	6	7
55. Sometimes I'm not aware that I'm smoking.	1	2	3	4	5	6	7
56. I'm worried that if I quit smoking I'll gain weight.	1	2	3	4	5	6	7
57. Smoking helps me think better.	1	2	3	4	5	6	7
58. Smoking really helps me feel better if I've been feeling down.	1	2	3	4	5	6	7
59. Some things are very hard to do without smoking.	1	2	3	4	5	6	7
60. Smoking makes me feel good.	1	2	3	4	5	6	7
61. Smoking keeps me from overeating.	1	2	3	4	5	6	7
62. My smoking is out of control.	1	2	3	4	5	6	7
63. I consider myself a heavy smoker.	1	2	3	4	5	6	7
64. Even when I feel good, smoking helps me feel better.	1	2	3	4	5	6	7
65. I reach for cigarettes when I feel irritable.	1	2	3	4	5	6	7
66. I enjoy the sensations of a long, slow exhalation of smoke.	1	2	3	4	5	6	7
67. Giving up cigarettes would be like losing a good friend.	1	2	3	4	5	6	7
68. Smoking is the easiest way to give myself a lift.	1	2	3	4	5	6	7

Fagerström Test for Nicotine Dependence (Heatherton et al., 1991)

Please answer the following questions:

	0	1	2	3
1. How soon after you wake up do you smoke your first cigarette?	After 60 Minutes	31 – 60 minutes	6-30 minutes	Within 5 minutes
2. Do you find it difficult to refrain from smoking in places where it is forbidden, e.g., in church, at the library, cinema, etc?	No	Yes		
3. Which cigarette would you hate most to give up?	All others	The first one in the morning		
4. How many cigarettes/day do you smoke?	10 or less	11-20	21-30	31 or more
5. Do you smoke more frequently during the first hours of waking than during the rest of the day?	No	Yes		
6. Do you smoke if you are so ill that you are in bed most of the day?	No	Yes		

Questionnaire for Smoking Urges (Cox et al., 2001)

Instructions: Indicate how much you agree or disagree with each of the following statements by circling the number between strongly disagree and strongly agree. The closer you choose a number to one end or the other indicates the strength of your disagreement or agreement. Please complete every item. We are interested in how you are thinking or feeling **right now** as you are filling out the questionnaire.

	Strongly Disagree					**Strongly Agree**
1. I have a desire for a cigarette.	0	1	2	3	4	5
2. Nothing would be better than smoking a cigarette**.**	0	1	2	3	4	5
3. If it were possible, I probably would smoke a cigarette.	0	1	2	3	4	5
4. I would control things better if I could smoke**.**	0	1	2	3	4	5
5. All I want is a cigarette.	0	1	2	3	4	5
6. I have an urge for a cigarette.	0	1	2	3	4	5
7. A cigarette would taste good**.**	0	1	2	3	4	5
8. I would do almost anything for a cigarette.	0	1	2	3	4	5
9. Smoking would make me less depressed.	0	1	2	3	4	5
10. I am going to smoke as soon as possible.	0	1	2	3	4	5

Assessment of Tobacco Exposure & Smoking on PDA (Author Constructed)

Questions	**Response Options**				
How many advertisements for cigarettes have you seen?	None	1	2	3	4 or more
Did you see any advertisements for menthol cigarettes?	Yes, I saw a menthol ad	I saw a non-menthol ad	I did not see any ads		
Did you see advertisements in a convenience or grocery store?	Yes	No	I did not see any ads		
Did you see advertisements in a bar or restaurant?	Yes	No	I did not see any ads		
Did you see advertisements on the internet?	Yes	I saw an ad somewhere else	I did not see any ads		
Did you see a Newport advertisement?	Yes	No	I did not see any ads		
Did you see a Kool advertisement?	Yes	No	I did not see any ads		
Did you see a Marlboro advertisement?	Yes	I saw ads for other brands	I did not see any ads		
Have you purchased any cigarettes at all?	Yes	No			
Have you purchased any cigarettes "on impulse"?	Yes	No			
How many cigarettes have you smoked?	None	1 cigarette	2 cigarettes	3 cigarettes	4 or more cigarettes

Self-report Attentional Bias Questionnaire (Leventhal et al., 2007)

For each of the following items, please circle a number that best describes you.

- **So far today, how often have you found your attention drawn to cigarettes?**

0	1	2	3	4
Not at all	A little bit	A moderate amount	A lot	An extreme amount

- **So far today, how attractive have you found the sight of cigarettes?**

0	1	2	3	4
Not at all attractive	A little attractive	Moderately attractive	Very attractive	Extremely attractive

- **So far today, how sensitive have you been to the smell of smoke?**

0	1	2	3	4
Not at all sensitive	A little sensitive	Moderately sensitive	Very sensitive	Extremely sensitive

- **So far today, how attractive have you found the smell of smoke?**

0	1	2	3	4
Not at all attractive	A little attractive	Moderately attractive	Very attractive	Extremely attractive

- **So far today, how often have you found yourself noticing people smoking?**

0	1	2	3	4
Not at all	A little bit	A moderate amount	A lot	An extreme amount

- **So far today, how often have you found yourself staring at cigarettes and cigarette smoke?**

0	1	2	3	4
Not at all	A little bit	A moderate amount	A lot	An extreme amount

- **So far today, how often have thoughts or images of smoking popped into you mind?**

0	1	2	3	4
Not at all	A little bit	A moderate amount	A lot	An extreme amount

- **So far today, how attractive have thoughts or images of smoking been?**

0	1	2	3	4
Not at all attractive	A little attractive	Moderately attractive	Very attractive	Extremely attractive

Eyetracker Questions (Author Constructed)

1. **Which advertisement was more attractive to you?**
    1. Ad 1
    2. Ad 2
    3. Don't Know
    4. Refuse to Answer
    5. Not Applicable

2. **What product was advertised in the first ad?**

3. **What product was advertised in the second ad?**

4. **What brand was advertised in the first ad?**

5. **What brand was advertised in the second ad?**

Smoking Diary (Author Constructed)

Study ID:_____

Tobacco Use Record Form

Instructions for Participant:

- **Complete this form each day.**
- **Just before going to sleep, indicate how many cigarettes you have smoked that day.**
- **Be honest… Accurate information is important!**

I agree to complete this form every night. I will provide information that is as accurate as possible.

SIGNATURE AND DATE:

	Monday	Tuesday	Wednesday	Thursday	Friday	Saturday	Sunday
Week 1							
Week 2							
Week 3							

Post-treatment Questionnaire (Author Constructed)

1. Did you notice any pattern in how the dots replaced certain pictures?

PTIQ_1 Did you notice any pattern in how the dots replaced certain pictures?

0 = No
1 = Yes

2. **Please provide a description of what you noticed:**

PTIQ_1B Please provide a description of what you noticed:

3. **There were two treatment conditions in this study. In the active condition the experimenter was trying to manipulate your attention so that your craving for cigarettes would be reduced. In the control (or inactive) condition there was no attempt to manipulate your attention. You were assigned to one of these two treatment conditions. Which condition do you think you were in?**

PTIQ_2 Which condition do you think you were in?

0 = Inactive
1 = Active

4. **Did you find the intervention to be acceptable?**

PTIQ_3 Did you find the intervention to be acceptable?

0 = No
1 = Yes

5. **How likely are you to recommend this treatment to a friend?**

PTIQ_4 How likely are you to recommend this treatment to a friend?

0 = Definitely not
1 = Possibly not
2 = Possibly
3 = Probably
4 = Definitely

6. **How boring/interesting was the intervention?**

PTIQ_5 How boring/interesting was the intervention?

0 = Very boring
1 = Somewhat boring
2 = Not boring
3 = Interesting
4 = Very interesting

7. **Do you plan on quitting in the next 30 days?**

PTIQ_6 Do you plan on quitting in the next 30 days?

0 = Definitely not
1 = Possibly not
2 = Possibly
3 = Probably
4=Definitely

Carbon Monoxide Assessment (Bedfont Micro Smokerlyzer, Harrietsham, England)

English

Micro⁺ Operating Manual

Quick Start Guide

1. Press the on/off button until the display becomes active. Release the button.

2. Insert the D-piece into the instrument and a new Steribreath™ mouthpiece

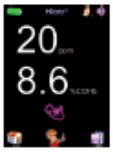
3. Touch the icon to start a breath test.

4. This starts the breath-hold countdown. The patient should inhale deeply and hold their breath while the display counts down to zero. If unable to hold their breath for the full countdown, see Warnings on page 4 or Settings on page 12.

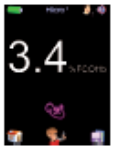
5. The audio bleep will sound during the last three seconds of the countdown.

6. At end of the countdown, the patient should blow slowly into the mouthpiece, and exhale until their lungs are empty.

7. The ppm and %COHb value will rise, and the highest level will hold.



8. To view the corresponding %FCOHb, touch the .



9. Remove and dispose of the Steribreath™ mouthpieces.

10. Remove the D-piece between tests to allow fresh air to purge sensor.

11. Touch to perform another breath test. A new mouthpiece is required.

12. To switch off, press the on/off button for 3 seconds. Unit will also auto power-off after 5 minutes of inactivity.

Cotinine Assay (Salimetrics LLC, State College, PA)

Cotinine Determination
P/N: 1-2002 research kit; P/N: 1-2112 diagnostic kit

All samples will be tested for salivary cotinine using a highly-sensitive enzyme immunoassay (Salimetrics LLC, State College, PA). The test will use a 20 µl of saliva sample per determination, with a lower limit of sensitivity of 0.15 ng/mL, a range of standard cur from 0.8 to 200 ng/mL, an average intra-assay coefficient of variation of 6.4 % and an average inter-assay coefficient of variation of 6.6 %.

SALIMETRICS COTININE EIA ASSAY
PERFORMANCE CHARACTERISTICS

A. **RECOVERY**:
Three saliva samples were spiked with known quantities of cotinine and assayed.

SAMPLE	ENDOGENOUS (ng/ml)	ADDED (ng/ml)	EXPECTED (ng/ml)	OBSERVED (ng/ml)	RECOVERY (%)
1	3.26	5	8.26	8.22	99.6
1	2.96	50	52.96	60.45	114.1
1	3.22	100	103.22	102.27	99.1
2	0.00	500	500.00	470.32	94.1
3	17.02	5	22.02	20.69	94.0
3	17.02	50	67.02	64.77	96.6

B. **PRECISION**:
The intra-assay precision was determined from 10 samples each of four levels of cotinine.

	N	MEAN (ng/mL)	Std Dev (ng/mL)	COEFFICIENT OF VARIATION (%)
1	10	5.49	0.25	4.5
2	10	52.35	4.50	8.6
3	10	105.21	6.16	5.9
4	10	495.47	32.04	6.5

The inter-assay precision was determined from the mean of average duplicates for 8 separate runs.

SAMPLE	N	MEAN (ng/mL)	Std Dev (ng/mL)	COEFFICIENT OF VARIATION (%)
low	8	6.07	0.55	9.04
high	8	102.23	4.30	4.21

C. **SENSITIVITY**:

The lower limit of sensitivity was determined by interpolating the mean optical density minus 2 SDs of 10 sets of duplicates at the 0 ng/mL level. The minimal concentration of cotinine that can be distinguished from 0 (zero) is 0.15 ng/mL.

ILUTION:
mples were diluted with assay diluent and assayed.

DILUTION FACTOR	EXPECTED (ng/mL)	OBSERVED (ng/mL)	RECOVERY (%)
		120.27	
1:2	60.14	56.87	94.6
1:4	30.07	27.12	90.2
1:8	15.03	14.00	93.1
1:16	7.52	6.77	90.0
1:32	3.76	3.50	93.2
1:64	1.88	1.82	96.7
		538.34	
1:2	269.17	278.40	103.4
1:4	134.59	139.68	103.8
1:8	67.29	67.27	100.0
1:16	33.65	33.34	99.1
1:32	16.82	16.91	100.5
1:64	8.41	9.24	109.8

SALIMETRICS®
101 Innovation Boulevard, Suite 302, State College, PA 16803 • USA
Tel 814.234.7748 • Fax 814.234.1608 • www.salimetrics.com

Saliva Sample Handling, Security & Data Reporting

Overview

Frozen saliva samples are shipped to Salimetrics LLC (State College, PA) on dry ice via overnight priority shipping. Samples are logged into the laboratory's database upon receipt, labeled with unique barcoded number (Salimetrics ID) and held at -20°C until testing commences. (Average testing turnaround time is 4-6 weeks). No human derived specimens will be accepted from any researcher or practitioner for testing, with individual personal patient identification information, including but not limited to patient name, social security number or address. At no time in the testing process are samples identified by name of subject or any information that would link the sample directly to an individual.

Sample Handling

Prior to analysis, samples are thawed at room temperature (20 to 22°C), centrifuged (1500 x g) for 15 minutes and assayed per researcher's instructions. Samples are returned to the freezer upon completion of pipetting. Assay data is reviewed by the supervisor and samples needing to be retested are identified. Samples needing retesting are again thawed, analyzed and refrozen.

Data Reporting

A preliminary data report is compiled and verified by the laboratory supervisor. Information from the original sample roster (timepoint information, dates of collection, etc.) is added to the report at this time. The technical supervisor reviews the preliminary report and generates the final report. The final report in Excel format is emailed to the investigator. A paper copy is mailed only if specifically requested.

Security

All data produced is secured to prevent alteration. Laboratory staff have exclusive secure access to save study data, and generate preliminary and final reports. All other Salimetrics personnel are barred from using or viewing these data files. Data file

Appendix B. Informed Consent and IRB Approval

UNIFORMED SERVICES UNIVERSITY OF THE HEALTH SCIENCES
4301 JONES BRIDGE ROAD
BETHESDA, MARYLAND 20814-4712
http://www.usuhs.mil

UNIFORMED SERVICES UNIVERSITY
BETHESDA, MARYLAND

This consent form is valid only if it contains the IRB stamped date

Consent for Voluntary Participation in a Non-Clinical Research Study

1. INTRODUCTION OF THE STUDY

You are being asked to be in a research study called "A Mobile Device-based Intervention to Reduce the Influence of Smoking Cues among African American Smokers" at the Uniformed Services University of the Health Sciences (USUHS), Bethesda, Maryland. You have been asked to take part in this study because you are a smoker. Participation in this research study is your choice. If you do not participate you will not be punished or lose access to benefits to which you are otherwise permitted. Please read the information below, and ask questions about anything you do not understand, before deciding whether to take part in the study.

2. PURPOSE OF THE STUDY

The purpose of this behavioral research study is to evaluate a new method of influencing smokers' attention, cravings and smoking. Results from this study may help researchers create more effective cessation (quitting) programs in the future. If you agree to be part of the study, you will be randomly assigned to one of two training conditions (i.e., you will be required to do one of two tasks on a mobile device). You will not know which condition you are in. This is the normal procedure in this type of study. In previous research by other investigators, the attention training has been delivered on a desktop computer in a laboratory setting. This research has shown that these two conditions can influence smokers' attention, cravings, and smoking differently. In this study, we want to see if we can deliver the training effectively on a mobile device (smartphone or PDA).

3. PROCEDURES TO BE FOLLOWED

You will attend up to 3 laboratory sessions in Building 28 at USUHS. The first laboratory session will last about 90 minutes and the second and third laboratory sessions will last about 60 minutes. You will first attend an orientation session (this session). At the orientation we will assess your color vision. The study requires that you are able to tell different colors apart, and so you will not be eligible if you are unable do this. If the investigators find that you are ineligible, your participation in the study will be over, and you will receive compensation for your time. If you are eligible and you agree to be in this study, a research staff member will show you how to use the mobile device. You will also complete a practice assessment on the mobile device. You will be asked to complete some brief questionnaires assessing your demographics (such as your age and income), your craving for cigarettes, and your smoking.

You will be asked to carry a mobile device around with you for 1 week. You can smoke as much or as little as you like during the week. The mobile device will beep you at random times during the day (about 4 times each day). After the mobile device beeps you, you will be asked to respond to a series of questions which ask you how you are feeling at that time, and ask you about your smoking. You will also perform a reaction time task on the PDA. Each mobile device assessment should last about 10 minutes in total. You will also record the number of cigarettes you smoke each day on a smoking diary.

You will be asked to attend a second session at USUHS Building 28. During this session you will complete reaction time tasks. You will then carry the mobile device for an additional, second, week. As before, during the week you will be prompted to complete a reaction time task and answer questions. Each assessment should last about 10 minutes in total.

You will attend a final laboratory session. During this session you will complete reaction time tasks and questionnaires in the laboratory. Following these task you will be asked to return the mobile device.

At the orientation, second session, and third session, you will be asked to provide a breath sample and a saliva sample. The breath sample and the saliva sample will help the researchers find out how much you have smoked. At the orientation session, the level of carbon monoxide in your breath must be above a certain level in order for you to be eligible for the study. Your craving for cigarettes will also be assessed at all laboratory visits.

At the second and third laboratory session you will complete a 5-minute task that examines how you look at a picture. To complete this assessment you will need to wear a small lightweight headset that allows us to measure where you are looking.

When your participation in the study is over, you will be offered self-help materials for quitting smoking and a referral to smoking cessation programs.

4. NUMBER OF PEOPLE THAT WILL TAKE PART IN THIS STUDY

Up to 80 individuals (including individuals who are ineligible) are expected to participate in this study.

5. AMOUNT OF TIME FOR YOU TO COMPLETE THE STUDY

Participation in this study will require in total about 11 and a half hours of your time over a period of about 2 weeks.

6. ELIGIBILITY AND PAYMENT FOR BEING IN THIS STUDY

Participation:

Civilians and military personnel may participate in this study. Federal civilians and military personnel must provide the investigators with a signed Statement of Approval form.

_____Participant Initial
_____Date

Compensation:

Civilians may receive compensation for their participation in this study. Military personnel and Federal civilians can be compensated during non-duty hours.

Non-federal civilians will receive $20 for completing the orientation session (even if ineligible), and $20 for completing the second laboratory and third laboratory session. Non-federal civilians will receive $1 for each mobile device assessment that they complete. They will also receive $3 for each day (except the final day) that they contribute data to the study, up to a maximum of 14 days. If a non-federal civilian completes 80% of scheduled mobile device assessments, they will receive approximately $146.80 [$60 (3 laboratory sessions; 3 sessions x $20) + $42 (days completed in study; 14 days x $3) + $44.80 (mobile device assessments; 4 Random assessment s x 14 days x $1 x .80)].

Federal civilians and Military will only receive compensation for the laboratory sessions and the mobile device assessments that occur during non-duty hours. For example, if a federal civilian or military member completes the orientation and second laboratory session during non-duty hours, and completes 1 mobile device assessment per day during non-duty hours, they will receive $116 ($60 (laboratory sessions; 3 sessions x $20) + $42 (days completed in study;14 days X $3) + $14 (1 Random Assessments x 14 days x $1).

Participants will receive payment in cash at the study visits.

7. POSSIBLE RISKS OR DISCOMFORTS FROM BEING IN THIS STUDY

The risks or discomforts from being in this study are expected to be minimal. There are no known risks associated with completing the laboratory assessments or the mobile device assessments. There is no reason to believe that your smoking will be increased by participation in the study.

You may refuse to answer any question that makes you feel uncomfortable. If you have concerns after completing the questionnaires, you are encouraged to contact your doctor or the study investigator. **Please do not respond to the mobile device while operating a vehicle because it is dangerous and may be illegal. If you do respond to the device while driving we are not responsible for accidents or tickets.** If something in this research makes you uncomfortable or upset, you may choose to stop taking part in this research at any time without loss of benefits; you may contact the investigator for referral. If the investigators note any distress or anxiety associated with the research, you will receive referrals, if appropriate.

8. POSSIBLE BENEFITS FROM BEING IN THIS STUDY

Some participants may reduce their smoking over the course of the week. Some participants may experience reduced cravings. However, no benefit can be guaranteed.

The information we learn may help us develop better programs to help people quit smoking. Therefore,

_____Participant Initial
_____Date

smokers may benefit from what is learned. This may be beneficial to society.

9. CONFIDENTIALITY/PRIVACY AND HOW YOUR IDENTITY AND YOUR RESEARCH RECORDS WILL BE MAINTAINED

All information you provide as part of this study will be confidential and will be protected to the fullest extent provided by law. Your responses to our laboratory and mobile device assessments will be maintained in a locked filing cabinet or on a password-protected computer in lab offices in the Department of Medical and Clinical Psychology. All records related to this study will be accessible to those persons directly involved in conducting this study and members of the USUHS Institutional Review Board (IRB), which provide oversight for protection of human research volunteers. In addition, the IRB at USUHS and other federal agencies that help protect people who are involved in research studies, may need to see the information you give us. Other than those groups, records from this study will be kept private to the fullest extent of the law. Scientific reports that come out of this study will not use your name or identify you in any way.

The breath sample you provide will allow us to measure carbon monoxide (CO) levels in your breath. This will allow us to measure how much you have smoked. We will use a standard CO monitor according to the manufacturer's instructions. Data on your CO levels will be stored on a password-protected EXCEL spreadsheet on a computer in Room 113 of Building 28. The password is only known to the research staff.

To prepare for the saliva sample, you will be asked to refrain from eating and drinking for 10 minutes before sampling. You will be offered a moist towelette to clean/wipe your hands/mouth. Using gloves, the research assistant will open the vial and give you the cotton roll. You will be asked to place the cotton piece in your mouth and to gently roll it in your mouth for a whole minute to saturate with saliva. You are requested to place most of the cotton piece on the edge of your mouth and re-insert it to the vial without touching the vial. Using gloves, the research assistant will tightly replace the cap on the vial.

The saliva samples will be stored in a freezer (-80F) in Building 28 for up to three months. Batches of saliva samples will be sent to Salimetrics, Inc. (www.salimetrics.com). Salimetrics, LLC, will perform an assay (a test) on each sample to determine the level of cotinine in the saliva. Cotinine is a breakdown product of nicotine and tells us how much you smoked during the past few days. No other tests will be performed on the saliva samples.

Only the study researchers will have access to the saliva samples. The samples are labeled with the participant study number (and visit number); only the research staff know the linkages between study numbers and participants. Thus, confidentiality is maintained during storage and distribution. The shipping procedures follow the U.S. Centers for Disease Control (CDC) guidelines for transport of biological specimens. Once the cotinine assay is performed, Salimetrics, LLC will destroy the samples. Because you are free to drop out of the study at any time, you can request that your saliva samples are destroyed. Saliva samples will only be stored at USUHS.

_____Participant Initial
_____Date

10. CONDITIONS WHICH YOUR PARTICIPATION IN THIS STUDY MAY BE STOPPED WITHOUT YOUR CONSENT

The investigator may stop you from participating if you experience difficulty in following the procedures. The investigator may also stop you participating if the mobile deviceis lost, stolen, or damaged.

11. IF YOU DECIDE TO STOP TAKING PART IN THIS STUDY AND THE INSTRUCTIONS FOR STOPPING EARLY

You have the right to withdraw from this study at any time. If you decide to stop taking part in this study, you should tell the principal investigator as soon as possible.

12. RECOURSE IN THE EVENT OF INJURY

If at any time you believe you have suffered an injury or illness as a result of participating in this research project, you should contact the Director of Human Research Protections Programs at the Uniformed Services University of the Health Sciences, Bethesda, Maryland 20814-4799 at (301) 295-9534. This office can review the matter with you, can provide information about your rights as a subject, and may be able to identify resources available to you. If you believe the government or one of the government's employees (such as a military doctor) has injured you, a claim for damages (money) against the federal government (including the military) may be filed under the Federal Torts Claims Act. Information about judicial avenues of compensation is available from the University's General Counsel at (301) 295-3028.

CONTACT FOR QUESTIONS OR PROBLEMS

If you have questions about this research, you should contact Cendrine Robinson or Dr. Andrew Waters, the individuals in charge of the study. Cendrine's number at USUHS is 301 295-1535 and Dr. Waters can be reached at 301 295-9675. During the evenings or on weekends, you can leave a message at that number. If you have questions about your rights as a research subject, you should call the Director of Human Research Protections Programs at USUHS at 301 295-9534. She is your representative and has no connection to the researcher conducting this study.

SIGNATURE OF RESEARCH PARTICIPANT OR LEGAL REPRESENTATIVE

You have read (or someone has read to you) the information in this consent form. You have been given a chance to ask questions and all of your questions have been answered to your satisfaction.

BY SIGNING THIS CONSENT FORM, YOU FREELY AGREE TO TAKE PART IN THE RESEARCH IT DESCRIBES.

_____ _____

Participant's Signature Date

_____

Participant's Printed Name

V.4 8-8-13

_____ Participant Initial

_____ Date

USUHS IRB APPROVED
From: 23 Aug 2013
Expires: 17 Aug 2014

SIGNATURE OF INVESTIGATOR/RESEARCH TEAM MEMBER

You have explained the research to the participant, or his/her legal representative, and answered all of his/her questions. You believe that the volunteer subject understands the information described in this document and freely consents to participate.

_____

Investigator's/Research Team Member's Signature Date (must be the same as the participant's)

_____

Investigator's/ Research Team Member's Printed Name

SIGNATURE OF WITNESS

Your signature as witness is intended to attest that the information in the consent document and any other information was explained to and apparently understood by the participant, or the participant's legal representative, that questions and concerns were addressed and that informed consent was freely given.

_____

Witness' Signature Date (must be the same as the participant's)

_____

Witness' Printed Name

USUHS IRB APPROVED
23 Aug 2013
Expires: 17 Dec 2014

_____ Participant Initial

_____ Date

UNIFORMED SERVICES UNIVERSITY OF THE HEALTH SCIENCES
4301 JONES BRIDGE ROAD
BETHESDA, MARYLAND 20814-4799

June 18, 2013

MEMORANDUM FOR CENDRINE ROBINSON, DEPARTMENT OF MEDICAL AND CLINICAL PSYCHOLOGY

SUBJECT: USUHS IRB #1 (FWA 00001628; DoD Assurance P60001) Approval of (TO-MPS-72-2418) for Human Subjects Participation

Congratulations! The Initial Review for your No More Than Minimal Risk human subjects research protocol TO-MPS-72-2418, entitled "A Smartphone-based Intervention to Reduce the Influence of smoking Cues among African American Smokers," was reviewed and approved for execution on June 18, 2013 by Edmund Howe, M.D., J.D. under the provisions of 32 CFR 219.110(b)(1)Suppl. F(6) and 32 CFR 219.110(b)(1)Suppl. F(7). This approval will be reported to the USU IRB #1 scheduled to meet on July 11, 2013.

The purpose of this study is to investigate the efficacy of Attentional Retraining in African American smokers using a modified dot probe task.

Authorization to conduct protocol TO-MPS-72-2418 will automatically terminate on June 17, 2014. If you plan to continue data collection or analysis beyond this date, IRB approval for continuation is required. Please submit a USU Form 3204 A/B, application for continuing approval 60 days prior to your termination date. You will receive a reminder from IRBNet.

You are required to submit amendments to this protocol, changes to the informed consent document (if applicable), adverse event reports, and other information pertinent to human research for this project in IRBNet. No changes to this protocol may be implemented prior to IRB approval. If you have questions regarding this IRB action or questions of a more general nature concerning human participation in research, please contact Trish Healy at 301-295-3388 or patricia.healy@usuhs.edu.

Edmund G. Howe, M.D., J.D.
Chair, IRB #1

Concur / Nonconcur

John E. McManigle, M.D.
Dean, School of Medicine (Acting)

This document has been signed electronically.

"Electronic Signature Notice: In accordance with the "Government Paperwork Elimination Act" (GPEA) (Pub.L. 105-277; codified at 44 USC 3504); Federal and DOD applicable instructions, directives and regulations, documents have been electronically signed and authorized by all who have been required to do so. These signatures have the same effect as their paper-based counterparts. Verification is retained within our protected electronic records and audit trails."

Appendix C. Flyer

Appendix D. Pictures for PDA Field Assessment and Mobile Eye

Pictures for PDA field assessments

Non Smoking Object

Smoking Object

Non Smoking Human

Smoking Human

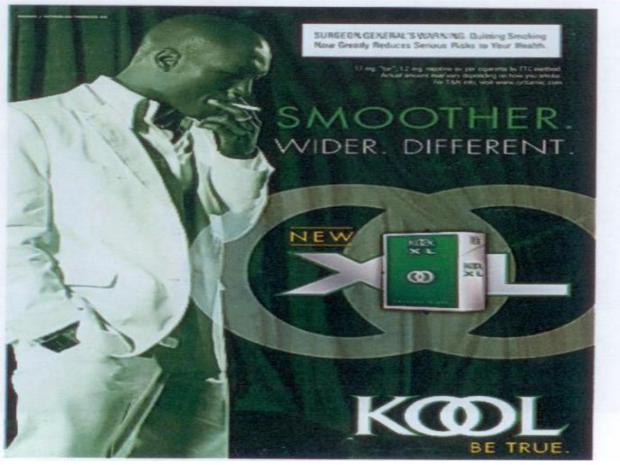
Cued Craving Pictures

Example Mobile Eye Picture

Training Pictures for Self-reported exposure to cues

In Store

Magazine



Outdoors

REFERENCES

1. APA. 2013. *Diagnostic and statistical manual of mental disorders, (DSM-5®)*. American Psychiatric Pub

2. National Institutes of Health. 2003. National Institutes of Health (NIH) strategic research plan to eliminate health disparities;. National Institutes of Health

3. Adams CD. 1982. Variations in the sensitivity of instrumental responding to reinforcer devaluation. *The Quarterly Journal of Experimental Psychology* 34:77-98

4. Agaku IT, King BA, Dube SR, Control CfD, Prevention. 2014. Current cigarette smoking among adults—United States, 2005–2012. *MMWR Morb Mortal Wkly Rep* 63:29-34

5. Ahluwalia JS, Harris KJ, Catley D, Okuyemi KS, Mayo MS. 2002. Sustained-release bupropion for smoking cessation in African Americans: a randomized controlled trial. *JAMA* 288:468-74

6. Ahluwalia JS, Okuyemi K, Nollen N, Choi WS, Kaur H, et al. 2006. The effects of nicotine gum and counseling among African American light smokers: a 2× 2 factorial design. *Addiction* 101:883-91

7. American Psychiatric Association. 2000. *Diagnostic and statistical manual of mental disorders, text revision (DSM-IV-TR)*. American Psychiatric Association

8. Ammerman A, Corbie-Smith G, St. George DMM, Washington C, Weathers B, Jackson-Christian B. 2003. Research expectations among African American church leaders in the PRAISE! project: a randomized trial guided by community-based participatory research. *American Journal of Public Health* 93:1720-7

9. Anderson SJ. 2011. Marketing of menthol cigarettes and consumer perceptions: a review of tobacco industry documents. *Tobacco Control* 20:ii20-ii8

10. Arnett JJ, Terhanian G. 1998. Adolescents' responses to cigarette advertisements: links between exposure, liking, and the appeal of smoking. *Tobacco Control* 7:129-33

11. Attwood AS, O'Sullivan H, Leonards U, Mackintosh B, Munafò MR. 2008. Attentional bias training and cue reactivity in cigarette smokers. *Addiction* 103:1875-82

12. Bar‐Haim Y. 2010. Research review: attention bias modification (ABM): a novel treatment for anxiety disorders. *Journal of Child Psychology and Psychiatry* 51:859-70

13. Barbeau EM, Wolin KY, Naumova EN, Balbach E. 2005. Tobacco advertising in communities: associations with race and class. *Preventive Medicine* 40:16-22

14. Beard C, Sawyer AT, Hofmann SG. 2012. Efficacy of attention bias modification using threat and appetitive stimuli: A meta-analytic review. *Behavior Therapy* 43:724-40

15. Benowitz N. 2008. Clinical pharmacology of nicotine: implications for understanding, preventing, and treating tobacco addiction. *Clinical Pharmacology & Therapeutics* 83:531-41

16. Benowitz NL. 1983. The use of biologic fluid samples in assessing tobacco smoke consumption. *NIDA Res Monogr* 48:6-26

17. Benowitz NL. 2001. Compensatory smoking of low-yield cigarettes. *National Cancer Institute. Risks associated with smoking cigarettes with low machine yields of tar and nicotine. Smoking and Tobacco Control Monograph No* 13:39-63

18. Benowitz NL. 2008. Neurobiology of nicotine addiction: implications for smoking cessation treatment. *The American Journal of Medicine* 121:S3-S10

19. Benowitz NL, Herrera B, Jacob P. 2004. Mentholated cigarette smoking inhibits nicotine metabolism. *Journal of Pharmacology and Experimental Therapeutics* 310:1208-15

20. Benowitz NL, Jacob P, Hall S, Tsoh J, Ahijevych K, et al. 2002. Biochemical verification of tobacco use and cessation. *Nicotine and Tobacco Research* 4:149-59

21. Berg CJ, Schauer GL, Ahluwalia JS, Benowitz NL. 2012. Correlates of NNAL levels among nondaily and daily smokers in the college student population. *Current Biomarker Findings* 2012

22. Berrendero F, Robledo P, Trigo JM, Martín-García E, Maldonado R. 2010. Neurobiological mechanisms involved in nicotine dependence and reward: participation of the endogenous opioid system. *Neuroscience & Biobehavioral Reviews* 35:220-31

23. Berridge KC. 2009. Wanting and Liking: Observations from the Neuroscience and Psychology Laboratory. *Inquiry (Oslo, Norway)* 52:378-

24. Braveman P. 2006. Health disparities and health equity: concepts and measurement. *Annu. Rev. Public Health* 27:167-94

25. Braveman P, Egerter S, Williams DR. 2011. The social determinants of health: coming of age. *Annual Review of Public Health* 32:381-98

26. Burton S, Clark L, Jackson K. 2012. The association between seeing retail displays of tobacco and tobacco smoking and purchase: findings from a diary‐style survey. *Addiction* 107:169-75

27. Caggiula AR, Donny EC, White AR, Chaudhri N, Booth S, et al. 2001. Cue dependency of nicotine self-administration and smoking. *Pharmacology Biochemistry and Behavior* 70:515-30

28. Caraballo RS, Giovino GA, Pechacek TF, Mowery PD, Richter PA, et al. 1998. Racial and ethnic differences in serum cotinine levels of cigarette smokers: Third National Health and Nutrition Examination Survey, 1988-1991. *JAMA* 280:135-9

29. Carpenter KM, Schreiber E, Church S, McDowell D. 2006. Drug Stroop performance: relationships with primary substance of use and treatment outcome in a drug-dependent outpatient sample. *Addictive Behaviors* 31:174-81

30. Carter-Pokras O, Baquet C. 2002. What is a" health disparity"? *Public health Reports* 117:426

31. Carter BL, Tiffany ST. 1999. Meta‐analysis of cue‐reactivity in addiction research. *Addiction* 94:327-40

32. Centers for Disease Control and Prevention. 2011. Vital signs: current cigarette smoking among adults aged≥ 18 years--United States, 2005-2010. *MMWR. Morbidity and mortality weekly report* 60:1207

33. Changeux J-P, Taly A. 2008. Nicotinic receptors, allosteric proteins and medicine. *Trends in Molecular Medicine* 14:93-102

34. Clark PI, Gautam S, Gerson LW. 1996. Effect of menthol cigarettes on biochemical markers of smoke exposure among black and white smokers. *CHEST Journal* 110:1194-8

35. Clattenburg EJ, Elf JL, Apelberg BJ. 2012. Unplanned cigarette purchases and tobacco point of sale advertising: a potential barrier to smoking cessation. *Tobacco Control*:tobaccocontrol-2012-050427

36. Coe JW, Brooks PR, Vetelino MG, Wirtz MC, Arnold EP, et al. 2005. Varenicline: an α4β2 nicotinic receptor partial agonist for smoking cessation. *Journal of Medicinal Chemistry* 48:3474-7

37. Cohen C, Perrault G, Griebel G, Soubrié P. 2005. Nicotine-associated cues maintain nicotine-seeking behavior in rats several weeks after nicotine withdrawal: reversal by the cannabinoid (CB1) receptor antagonist, rimonabant (SR141716). *Neuropsychopharmacology* 30:145-55

38. Cohen JD, Bartsch GE. 1980. A comparison between carboxyhemoglobin and serum thiocyanate determinations as indicators of cigarette smoking. *American Journal of Public Health* 70:284-6

39. Cokkinides VE, Halpern MT, Barbeau EM, Ward E, Thun MJ. 2008. Racial and ethnic disparities in smoking-cessation interventions: analysis of the 2005 National Health Interview Survey. *American Journal of Preventive Medicine* 34:404-12

40. Compton RJ, Banich MT, Mohanty A, Milham MP, Herrington J, et al. 2003. Paying attention to emotion. *Cognitive, Affective, & Behavioral Neuroscience* 3:81-96

41. Conklin CA, Perkins KA, Robin N, McClernon FJ, Salkeld RP. 2010. Bringing the real world into the laboratory: personal smoking and nonsmoking environments. *Drug and Alcohol Dependence* 111:58-63

42. Conklin CA, Robin N, Perkins KA, Salkeld RP, McClernon FJ. 2008. Proximal versus distal cues to smoke: the effects of environments on smokers' cue-reactivity. *Experimental and Clinical Psychopharmacology* 16:207

43. Covey LS, Botello-Harbaum M, Glassman AH, Masmela J, LoDuca C, et al. 2007. Smokers' response to combination bupropion, nicotine patch, and counseling treatment by race/ethnicity. *Ethnicity & Disease* 18:59-64

44. Cox LS, Tiffany ST, Christen AG. 2001. Evaluation of the brief questionnaire of smoking urges (QSU-brief) in laboratory and clinical settings. *Nicotine & Tobacco Research* 3:7-16

45. Cox WM, Fadardi JS, Klinger E. 2006. Motivational processes underlying implicit cognition in addiction. *Handbook of implicit cognition and addiction*:253-66

46. Cox WM, Hogan LM, Kristian MR, Race JH. 2002. Alcohol attentional bias as a predictor of alcohol abusers' treatment outcome. *Drug and Alcohol Dependence* 68:237-43

47. Dennis TA, O'Toole LJ. 2014. Mental Health on the Go Effects of a Gamified Attention-Bias Modification Mobile Application in Trait-Anxious Adults. *Clinical Psychological Science*:2167702614522228

48. Dickinson A, Wood N, Smith JW. 2002. Alcohol seeking by rats: action or habit? *The Quarterly Journal of Experimental Psychology: Section B* 55:331-48

49. DiFranza JR, Wellman RJ, Sargent JD, Weitzman M, Hipple BJ, Winickoff JP. 2006. Tobacco promotion and the initiation of tobacco use: assessing the evidence for causality. *Pediatrics* 117:e1237-e48

50. Do DP, Finch BK, Basurto-Davila R, Bird C, Escarce J, Lurie N. 2008. Does place explain racial health disparities? Quantifying the contribution of residential context to the Black/white health gap in the United States. *Social Science & Medicine* 67:1258-68

51. Eisenberg MJ, Filion KB, Yavin D, Bélisle P, Mottillo S, et al. 2008. Pharmacotherapies for smoking cessation: a meta-analysis of randomized controlled trials. *Canadian Medical Association Journal* 179:135-44

52. Enock PM, Hofmann SG, McNally RJ. 2014. Attention bias modification training via smartphone to reduce social anxiety: a randomized, controlled multi-session experiment. *Cognitive Therapy And Research* 38:200-16

53. Etter J-F. 2005. A comparison of the content-, construct-and predictive validity of the cigarette dependence scale and the Fagerström test for nicotine dependence. *Drug and Alcohol Dependence* 77:259-68

54. Etter J-F, Stapleton JA. 2006. Nicotine replacement therapy for long-term smoking cessation: a meta-analysis. *Tobacco Control* 15:280-5

55. Everitt BJ, Robbins TW. 2005. Neural systems of reinforcement for drug addiction: from actions to habits to compulsion. *Nature Neuroscience* 8:1481-9

56. Fadardi JS, Cox WM. 2009. Reversing the sequence: reducing alcohol consumption by overcoming alcohol attentional bias. *Drug and Alcohol Dependence* 101:137-45

57. Fagan P, King G, Lawrence D, Petrucci SA, Robinson RG, et al. 2004. Eliminating tobacco-related health disparities: directions for future research. *American Journal of Public Health* 94:211-7

58. Fagan P, Moolchan ET, Lawrence D, Fernander A, Ponder PK. 2007. Identifying health disparities across the tobacco continuum. *Addiction* 102:5-29

59. Fagerström K, Hughes J. 2008. Varenicline in the treatment of tobacco dependence. *Neuropsychiatric Disease And Treatment* 4:353

60. Federal Trade Commission. 2011. Cigarette Report for 2007 and 2008.

61. Ferguson SG, Shiffman S. 2009. The relevance and treatment of cue-induced cravings in tobacco dependence. *Journal of Substance Abuse Treatment* 36:235-43

62. Ferguson SG, Shiffman S, Gwaltney CJ. 2006. Does reducing withdrawal severity mediate nicotine patch efficacy? A randomized clinical trial. *Journal of consulting and Clinical Psychology* 74:1153

63. Field M, Cox WM. 2008. Attentional bias in addictive behaviors: a review of its development, causes, and consequences. *Drug and Alcohol Dependence* 97:1-20

64. Field M, Duka T, Eastwood B, Child R, Santarcangelo M, Gayton M. 2007. Experimental manipulation of attentional biases in heavy drinkers: do the effects generalise? *Psychopharmacology* 192:593-608

65. Field M, Duka T, Tyler E, Schoenmakers T. 2009. Attentional bias modification in tobacco smokers. *Nicotine & Tobacco Research* 11:812-22

66. Field M, Eastwood B. 2005. Experimental manipulation of attentional bias increases the motivation to drink alcohol. *Psychopharmacology* 183:350-7

67. Field M, Mogg K, Bradley BP. 2004. Eye movements to smoking-related cues: effects of nicotine deprivation. *Psychopharmacology* 173:116-23

68. Fiore M, Jaen CR, Baker T, Bailey W, Benowitz N, et al. 2008. Treating tobacco use and dependence: 2008 update. Rockville, MD: U.S. Department of Health and Human Services. Public Health Service.

69. Fitzmaurice GM, Laird NM, Ware JH. 2012. *Applied longitudinal analysis*. John Wiley & Sons

70. Fu SS, Kodl MM, Joseph AM, Hatsukami DK, Johnson EO, et al. 2008. Racial/ethnic disparities in the use of nicotine replacement therapy and quit ratios in lifetime smokers ages 25 to 44 years. *Cancer Epidemiology Biomarkers & Prevention* 17:1640-7

71. Fu SS, McFall M, Saxon AJ, Beckham JC, Carmody TP, et al. 2007. Post-traumatic stress disorder and smoking: a systematic review. *Nicotine & Tobacco Research* 9:1071-84

72. Fu SS, Sherman SE, Yano EM, van Ryn M, Lanto AB, Joseph AM. 2005. Ethnic disparities in the use of nicotine replacement therapy for smoking cessation in an equal access health care system. *American Journal of Health Promotion* 20:108-16

73. Gandhi KK, Foulds J, Steinberg M, Lu SE, Williams J. 2009. Lower quit rates among African American and Latino menthol cigarette smokers at a tobacco treatment clinic. *International Journal of Clinical Practice* 63:360-7

74. Gandini S, Botteri E, Iodice S, Boniol M, Lowenfels AB, et al. 2008. Tobacco smoking and cancer: A meta‐analysis. *International Journal Of Cancer* 122:155-64

75. Gardiner PS. 2004. The African Americanization of menthol cigarette use in the United States. *Nicotine & Tobacco Research* 6:S55-S65

76. George TP, O'Malley SS. 2004. Current pharmacological treatments for nicotine dependence. *Trends In Pharmacological Sciences* 25:42-8

77. Giovino GA, Sidney S, Gfroerer JC, O'Malley PM, Allen JA, et al. 2004. Epidemiology of menthol cigarette use. *Nicotine & Tobacco Research* 6:S67-S81

78. Goniewicz ML, Eisner MD, Lazcano-Ponce E, Zielinska-Danch W, Koszowski B, et al. 2011. Comparison of urine cotinine and the tobacco-specific nitrosamine metabolite 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanol (NNAL) and their ratio to discriminate active from passive smoking. *Nicotine & Tobacco Research*:ntq237

79. Gorber SC, Schofield-Hurwitz S, Hardt J, Levasseur G, Tremblay M. 2009. The accuracy of self-reported smoking: a systematic review of the relationship between self-reported and cotinine-assessed smoking status. *Nicotine & Tobacco Research* 11:12-24

80. Grunberg NE, & Starosciak, A. K. 2010. Nicotine. In *Encyclopedia of Behavioral Neuroscience*, ed. GF Koob, Le Moal, M., & Thompson R. F, 2:464-70: Oxford: Academic Press. Number of 464-70 pp.

81. Grunberg NE, Berger SS, Starosciak AK. 2011. 14 Tobacco Use. *Handbook of Health Psychology*:311

82. Haiman CA, Stram DO, Wilkens LR, Pike MC, Kolonel LN, et al. 2006. Ethnic and racial differences in the smoking-related risk of lung cancer. *New England Journal of Medicine* 354:333-42

83. Hakamata Y, Lissek S, Bar-Haim Y, Britton JC, Fox NA, et al. 2010. Attention bias modification treatment: a meta-analysis toward the establishment of novel treatment for anxiety. *Biological Psychiatry* 68:982-90

84. Haley NJ, Axelrad CM, Tilton KA. 1983. Validation of self-reported smoking behavior: biochemical analyses of cotinine and thiocyanate. *American Journal of Public Health* 73:1204-7

85. Hallion LS, Ruscio AM. 2011. A meta-analysis of the effect of cognitive bias modification on anxiety and depression. *Psychological Bulletin* 137:940

86. Hatsukami D, Hughes JR, Pickens R. 1985. Characterization of tobacco withdrawal: Physiological and subjective effects. In *Pharmacological Adjuncts In Smoking Cessation*, 53:56-67: National Institute on Drug Abuse Rockville, D. Number of 56-67 pp.

87. Heatherton TF, Kozlowski LT, Frecker RC, FAGERSTROM KO. 1991. The Fagerström test for nicotine dependence: a revision of the Fagerstrom Tolerance Questionnaire. *British Journal of Addiction* 86:1119-27

88. Henningfield JE, Goldberg SR. 1983. Nicotine as a reinforcer in human subjects and laboratory animals. *Pharmacology Biochemistry and Behavior* 19:989-92

89. Herning RI, Jones RT, Bachman J. 1983. EEG changes during tobacco withdrawal. *Psychophysiology* 20:507-12

90. Hoek J, Gifford H, Pirikahu G, Thomson G, Edwards R. 2010. How do tobacco retail displays affect cessation attempts? Findings from a qualitative study. *Tobacco Control* 19:334-7

91. Hogarth L, Chase HW, Baess K. 2012. Impaired goal-directed behavioural control in human impulsivity. *The Quarterly Journal of Experimental Psychology* 65:305-16

92. Hughes JR. 2007. Effects of abstinence from tobacco: valid symptoms and time course. *Nicotine & Tobacco Research* 9:315-27

93. Hughes JR, Gust, S. W., Skoog, K., & Keenan, R. M. 1991. Symptoms of tobacco withdrawal: A replication and extension. *Archives of General Psychiatry* 48:52-9

94. Hughes JR, Keenan RM, Yellin A. 1989. Effect of tobacco withdrawal on sustained attention. *Addictive Behaviors* 14:577-80

95. Hyland A, Garten S, Giovino G, Cummings KM. 2002. Mentholated cigarettes and smoking cessation: findings from COMMIT. *Tobacco Control* 11:135-9

96. Irwin A, Scali E. 2005. Action on the social determinants of health: learning from previous experiences. World Health Organization: Commission on Social Determinants of Health: Geneva.

97. Isaacs SL, Schroeder SA. 2004. Class-the ignored determinant of the nation's health. *New England Journal of Medicine* 351:1137-42

98. Israel BA, Schulz AJ, Parker EA, Becker AB. 1998. Review of community-based research: assessing partnership approaches to improve public health. *Annual Review of Public Health* 19:173-202

99. Janes AC, Pizzagalli DA, Richardt S, Chuzi S, Pachas G, et al. 2010. Brain reactivity to smoking cues prior to smoking cessation predicts ability to maintain tobacco abstinence. *Biological Psychiatry* 67:722-9

100. John R, Cheney MK, Azad MR. 2009. Point-of-sale marketing of tobacco products: taking advantage of the socially disadvantaged? *Journal of Health Care for the Poor And Underserved* 20:489-506

101. Jorde LB, Wooding SP. 2004. Genetic variation, classification and'race'. *Nature Genetics* 36:S28-S33

102. Jorenby DE, Hays JT, Rigotti NA, Azoulay S, Watsky EJ, et al. 2006. Efficacy of varenicline, an α4β2 nicotinic acetylcholine receptor partial agonist, vs placebo or sustained-release bupropion for smoking cessation: a randomized controlled trial. *JAMA* 296:56-63

103. Joseph AM, Hecht SS, Murphy SE, Carmella SG, Le CT, et al. 2005. Relationships between cigarette consumption and biomarkers of tobacco toxin exposure. *Cancer Epidemiology Biomarkers & Prevention* 14:2963-8

104. Kahneman D. 2003. A perspective on judgment and choice: mapping bounded rationality. *American Psychologist* 58:697

105. Kakoschke N, Kemps E, Tiggemann M. 2014. Attentional bias modification encourages healthy eating. *Eating Behaviors* 15:120-4

106. Keenan RM, Hatsukami DK, Pentel PR, Thompson TN, Grillo MA. 1994. Pharmacodynamic effects of cotinine in abstinent cigarette smokers. *Clinical Pharmacology & Therapeutics* 55:581-90

107. Kenny PJ, Markou A. 2001. Neurobiology of the nicotine withdrawal syndrome. *Pharmacology Biochemistry and Behavior* 70:531-49

108. Kerst WF. 2013. *Attenti*

on training in smokers. Uniformed Services University of the Health Sciences

109. Kerst WF, Waters AJ. 2014. Attentional retraining administered in the field reduces smokers' attentional bias and craving. *Health Psychology,* 33:1232-1240.

110. King G, Polednak A, Bendel RB, Vilsaint MC, Nahata SB. 2004. Disparities in smoking cessation between African Americans and whites: 1990-2000. *American Journal of Public Health* 94:1965-71

111. Kirchner TR, Cantrell J, Anesetti-Rothermel A, Ganz O, Vallone DM, Abrams DB. 2013. Geospatial exposure to point-of-sale tobacco: real-time craving and smoking-cessation outcomes. *American Journal of Preventive Medicine* 45:379-85

112. Knott VJ, Venables PH. 1977. EEG Alpha Correlates of Non‐Smokers, Smokers, Smoking, and Smoking Deprivation. *Psychophysiology* 14:150-6

113. Koob GF, Bloom FE. 1988. Cellular and molecular mechanisms of drug dependence. *Science* 242:715-23

114. Koob GF, Le Moal M. 1997. Drug abuse: hedonic homeostatic dysregulation. *Science* 278:52-8

115. Koob GF, Stinus L, Le Moal M, Bloom FE. 1989. Opponent process theory of motivation: neurobiological evidence from studies of opiate dependence. *Neuroscience & Biobehavioral Reviews* 13:135-40

116. Kreuter MW, McClure SM. 2004. The role of culture in health communication. *Annu. Rev. Public Health* 25:439-55

117. Kreuter MW, Skinner CS. 2000. Tailoring: what's in a name? *Health Education Research* 15:1-4

118. Kuckertz JM, Amir N, Boffa JW, Warren CK, Rindt SE, et al. 2014. The effectiveness of an attention bias modification program as an adjunctive treatment for Post-Traumatic Stress Disorder. *Behaviour Research And Therapy* 63:25-35

119. Lai DT, Cahill K, Qin Y, Tang JL. 2010. Motivational interviewing for smoking cessation. *The Cochrane Library*

120. Lancaster T, Stead LF. 2005. Individual behavioural counselling for smoking cessation. *The Cochrane Library*

121. LaVeist T, Pollack K, Thorpe R, Fesahazion R, Gaskin D. 2011. Place, not race: disparities dissipate in southwest Baltimore when blacks and whites live under similar conditions. *Health Affairs* 30:1880-7

122. LaVeist TA, Nickerson KJ, Bowie JV. 2000. Attitudes about racism, medical mistrust, and satisfaction with care among African American and white cardiac patients. *Medical Care Research and Review* 57:146-61

123. Laws MB, Whitman J, Bowser D, Krech L. 2002. Tobacco availability and point of sale marketing in demographically contrasting districts of Massachusetts. *Tobacco Control* 11:ii71-ii3

124. Leischow SJ, Ranger-Moore J, Lawrence D. 2000. Addressing social and cultural disparities in tobacco use. *Addictive Behaviors* 25:821-31

125. Leshner AI. 1997. Addiction is a brain disease, and it matters. *Science* 278:45-7

126. Leventhal AM, Waters AJ, Boyd S, Moolchan ET, Lerman C, Pickworth WB. 2007. Gender differences in acute tobacco withdrawal: effects on subjective, cognitive, and physiological measures. *Experimental and Clinical Psychopharmacology* 15:21

127. Leventhal AM, Waters AJ, Moolchan ET, Heishman SJ, Pickworth WB. 2010. A quantitative analysis of subjective, cognitive, and physiological manifestations of the acute tobacco abstinence syndrome. *Addictive Behaviors* 35:1120-30

128. Lichtenstein E, Zhu S-H, Tedeschi GJ. 2010. Smoking cessation quitlines: an underrecognized intervention success story. *American Psychologist* 65:252

129. Littell RC, Stroup WW, Milliken GA, Wolfinger RD, Schabenberger O. 2006. *SAS for mixed models*. SAS institute

130. Litvin EB, Brandon TH. 2010. Testing the influence of external and internal cues on smoking motivation using a community sample. *Experimental and Clinical Psychopharmacology* 18:61

131. Liu X, Caggiula AR, Yee SK, Nobuta H, Poland RE, Pechnick RN. 2006. Reinstatement of nicotine-seeking behavior by drug-associated stimuli after extinction in rats. *Psychopharmacology* 184:417-25

132. Lopes FM, Pires AV, Bizarro L. 2014. Attentional bias modification in smokers trying to quit: A longitudinal study about the effects of number of sessions. *Journal of Substance Abuse Treatment* 47:50-7

133. Lubman D, Peters L, Mogg K, Bradley B, Deakin J. 2000. Attentional bias for drug cues in opiate dependence. *Psychological Medicine* 30:169-75

134. MacLeod C, Koster EH, Fox E. 2009. Whither cognitive bias modification research? Commentary on the special section articles. *Journal of Abnormal Psychology* 118:89

135. MacLeod C, Mathews A, Tata P. 1986. Attentional bias in emotional disorders. *Journal of Abnormal Psychology* 95:15

136. MacLeod C, Rutherford E, Campbell L, Ebsworthy G, Holker L. 2002. Selective attention and emotional vulnerability: assessing the causal basis of their association through the experimental manipulation of attentional bias. *Journal of Abnormal Psychology* 111:107

137. Marhe R, Waters AJ, van de Wetering BJ, Franken IH. 2013. Implicit and explicit drug-related cognitions during detoxification treatment are associated with drug relapse: An ecological momentary assessment study. *Journal of consulting and clinical psychology* 81:1

138. Marissen MA, Franken IH, Waters AJ, Blanken P, Van Den Brink W, Hendriks VM. 2006. Attentional bias predicts heroin relapse following treatment. *Addiction* 101:1306-12

139. Markou A. 2008. Neurobiology of nicotine dependence. *Philosophical Transactions of the Royal Society B: Biological Sciences* 363:3159-68

140. Marteau TM, Hollands GJ, Fletcher PC. 2012. Changing human behavior to prevent disease: the importance of targeting automatic processes. *Science* 337:1492-5

141. Martino SC, Scharf DM, Setodji CM, Shadel WG. 2012. Measuring exposure to protobacco marketing and media: a field study using ecological momentary assessment. *Nicotine & Tobacco Research* 14:398-406

142. Matthews AK, Sánchez-Johnsen L, King A. 2009. Development of a culturally targeted smoking cessation intervention for African American smokers. *Journal of Community Health* 34:480-92

143. Mazas CA, Wetter DW. 2003. Smoking cessation interventions among African Americans: Research needs. *Cancer Control* 10:87-9

144. McAfee TA. 2007. Quitlines: A tool for research and dissemination of evidence-based cessation practices. *American Journal of Preventive Medicine* 33:S357-S67

145. McHugh RK, Murray HW, Hearon BA, Calkins AW, Otto MW. 2010. Attentional bias and craving in smokers: the impact of a single attentional training session. *Nicotine & Tobacco Research* 12:1261-4

146. Mihalak KB, Carroll FI, Luetje CW. 2006. Varenicline is a partial agonist at α4β2 and a full agonist at α7 neuronal nicotinic receptors. *Molecular Pharmacology* 70:801-5

147. Miles FJ, Everitt BJ, Dickinson A. 2003. Oral cocaine seeking by rats: action or habit? *Behavioral Neuroscience* 117:927

148. Millar NS, Gotti C. 2009. Diversity of vertebrate nicotinic acetylcholine receptors. *Neuropharmacology* 56:237-46

149. Mogg K, Bradley BP. 1998. A cognitive-motivational analysis of anxiety. *Behaviour Research And Therapy* 36:809-48

150. Mogg K, Bradley BP. 2002. Selective processing of smoking-related cues in smokers: manipulation of deprivation level and comparison of three measures of processing bias. *Journal of Psychopharmacology* 16:385-92

151. Mogg K, Bradley BP, Field M, De Houwer J. 2003. Eye movements to smoking‐related pictures in smokers: relationship between attentional biases and implicit and explicit measures of stimulus valence. *Addiction* 98:825-36

152. Mogoaşe C, David D, Koster EH. 2014. Clinical Efficacy of Attentional Bias Modification Procedures: An Updated Meta‐Analysis. *Journal of clinical Psychology* 70:1133-57

153. Moolchan ET, Fagan P, Fernander AF, Velicer WF, Hayward MD, et al. 2007. Addressing tobacco‐related health disparities. *Addiction* 102:30-42

154. Moore T. 1991. The African-American church: A source of empowerment, mutual help, and social change. *Prevention in Human Services* 10:147-67

155. Mottillo S, Filion KB, Bélisle P, Joseph L, Gervais A, et al. 2009. Behavioural interventions for smoking cessation: a meta-analysis of randomized controlled trials. *European Heart Journal* 30:718-30

156. Nakajima M, Fukami T, Yamanaka H, Higashi E, Sakai H, et al. 2006. Comprehensive evaluation of variability in nicotine metabolism and CYP2A6 polymorphic alleles in four ethnic populations. *Clinical Pharmacology & Therapeutics* 80:282-97

157. National Institute on Drug Abuse. 2014. In *The Science of Drug Abuse and Addiction: The Basics*

158. Niaura RS, Rohsenow DJ, Binkoff JA, Monti PM, Pedraza M, Abrams DB. 1988. Relevance of cue reactivity to understanding alcohol and smoking relapse. *Journal of Abnormal Psychology* 97:133

159. Nides M. 2008. Update on pharmacologic options for smoking cessation treatment. *The American Journal of Medicine* 121:S20-S31

160. Nielsen. 2012. America's new mobile majority: A look at Smartphone owners in the U.S. .

161. Office of Management and Budget. 2003. Revisions to the Standards for the Classification of Federal Data on Race and Ethnicity. ed. OoMa Budget

162. Okuyemi KS, Ebersole-Robinson M, Nazir N, Ahluwalia JS. 2004. African-American menthol and nonmenthol smokers: differences in smoking and cessation experiences. *Journal of the National Medical Association* 96:1208

163. Okuyemi KS, Pulvers KM, Cox LS, Thomas JL, Kaur H, et al. 2007. Nicotine dependence among African American light smokers: a comparison of three scales. *Addictive Behaviors* 32:1989-2002

164. Oncken C, Gonzales D, Nides M, Rennard S, Watsky E, et al. 2006. Efficacy and safety of the novel selective nicotinic acetylcholine receptor partial agonist, varenicline, for smoking cessation. *Archives of Internal Medicine* 166:1571-7

165. Ordoñana JR, González-Javier F, Gómez-Amor J. 2012. Psychophysiological reactivity to environmental tobacco smoke on smokers and non-smokers. *Addictive Behaviors* 37:838-43

166. Ossip-Klein DJ, Bigelow G, Parker SR, Curry S, Hall S, Kirkland S. 1986. Task Force 1: Classification and assessment of smoking behavior. *Health Psychology*

167. Patterson F, Benowitz N, Shields P, Kaufmann V, Jepson C, et al. 2003. Individual differences in nicotine intake per cigarette. *Cancer Epidemiology Biomarkers & Prevention* 12:468-71

168. Pelloux Y, Everitt BJ, Dickinson A. 2007. Compulsive drug seeking by rats under punishment: effects of drug taking history. *Psychopharmacology* 194:127-37

169. Perez-Stable EJ, Herrera B, Jacob III P, Benowitz NL. 1998. Nicotine metabolism and intake in black and white smokers. *JAMA* 280:152-6

170. Perkins KA. 2009. Does smoking cue‐induced craving tell us anything important about nicotine dependence? *Addiction* 104:1610-6

171. Peuker AC, Bizarro L. 2014. Attentional avoidance of smoking cues in former smokers. *Journal of Substance Abuse Treatment* 46:183-8

172. Pickworth WB, Herning RI, Henningfield JE. 1989. Spontaneous EEG changes during tobacco abstinence and nicotine substitution in human volunteers. *Journal of Pharmacology and Experimental Therapeutics* 251:976-82

173. Pierce RC, Vanderschuren LJ. 2010. Kicking the habit: the neural basis of ingrained behaviors in cocaine addiction. *Neuroscience & Biobehavioral Reviews* 35:212-9

174. Piper ME, Cook JW, Schlam TR, Jorenby DE, Smith SS, et al. 2010. Gender, race, and education differences in abstinence rates among participants in two randomized smoking cessation trials. *Nicotine & Tobacco Research*:ntq067

175. Piper ME, Piasecki TM, Federman EB, Bolt DM, Smith SS, et al. 2004. A multiple motives approach to tobacco dependence: the Wisconsin Inventory of Smoking Dependence Motives (WISDM-68). *Journal of Consulting And Clinical Psychology* 72:139

176. Polosa R, Benowitz NL. 2011. Treatment of nicotine addiction: present therapeutic options and pipeline developments. *Trends In Pharmacological Sciences* 32:281-9

177. Pomerleau CS, Carton SM, Lutzke ML, Flessland KA, Pomerleau OF. 1994. Reliability of the Fagerstrom tolerance questionnaire and the Fagerstrom test for nicotine dependence. *Addictive Behaviors* 19:33-9

178. Posner MI, Petersen SE. 1989. The attention system of the human brain, *Annual Review of Neuroscience* 13: 25-39

179. Powell J, Dawkins L, West R, Powell J, Pickering A. 2010. Relapse to smoking during unaided cessation: clinical, cognitive and motivational predictors. *Psychopharmacology* 212:537-49

180. Pradhan AK, Pollatsek A, Knodler M, Fisher DL. 2009. Can younger drivers be trained to scan for information that will reduce their risk in roadway traffic scenarios that are hard to identify as hazardous? *Ergonomics* 52:657-73

181. Primack BA, Bost JE, Land SR, Fine MJ. 2007. Volume of tobacco advertising in African American markets: systematic review and meta-analysis. *Public Health Reports* 122:607

182. Rabius V, Wiatrek D, McAlister AL. 2011. African American participation and success in telephone counseling for smoking cessation. *Nicotine & Tobacco Research* 14:240-242.

183. Reitzel LR, Cromley EK, Li Y, Cao Y, Mater RD, et al. 2011. The effect of tobacco outlet density and proximity on smoking cessation. *American Journal of Public Health* 101:315

184. Resnicow K, Soler R, Braithwaite RL, Ahluwalia JS, Butler J. 2000. Cultural sensitivity in substance use prevention. *Journal of* community psychology 28:271-90

185. Robinson CD, Pickworth WB, Heishman SJ, Waters AJ. 2014. The acute tobacco withdrawal syndrome among black smokers. *Psychology of Addictive Behaviors* 28:173

186. Robinson CD PW, Heishman SJ, Wetter DW, Cinciripini PM, Li Y, Rowell B, Waters AJ in press. African American cigarette smokers report more attention to smoking cues than White smokers: Implications for smoking cessation. *Nicotine Tob Res.*

187. Robinson JH, Pritchard WS. 1992. The meaning of addiction: reply to West. *Psychopharmacology* 108:411-6

188. Robinson TE, Berridge KC. 1993. The neural basis of drug craving: an incentive-sensitization theory of addiction. *Brain research reviews* 18:247-91

189. Robinson TE, Berridge KC. 2008. The incentive sensitization theory of addiction: some current issues. *Philosophical Transactions of the Royal Society B: Biological Sciences* 363:3137-46

190. Rodriguez D, Carlos HA, Adachi-Mejia AM, Berke EM, Sargent JD. 2012. Predictors of tobacco outlet density nationwide: a geographic analysis. *Tobacco Control*:tobaccocontrol-2011-050120

191. Rohsenow DJ, Monti PM. 1999. Does urge to drink predict relapse after treatment? *Alcohol Research and Health* 23:225-32

192. Rosse RB, Miller MW, Hess AL, Alim TN, Deutsch SI. 1993. Measures of visual scanning as a predictor of cocaine cravings and urges. *Biological Psychiatry* 33:554-6

193. Rudy JW, Stadler-Morris S, Albert P. 1987. Ontogeny of spatial navigation behaviors in the rat: dissociation of" proximal"-and" distal"-cue-based behaviors. *Behavioral Neuroscience* 101:62

194. Sarkar M, Wang J, Liang Q. 2012. Metabolism of Nicotine and 4-(methylnitrosamino)-l-(3-pyridyl)-lbutanone (NNK) in menthol and non-menthol cigarette smokers. *Drug Metabolism Letters* 6:198-206

195. Sasco A, Secretan M, Straif K. 2004. Tobacco smoking and cancer: a brief review of recent epidemiological evidence. *Lung Cancer* 45:S3-S9

196. Sastry BR, Chance M, Singh G, Horn J, Janson V. 1995. Distribution and retention of nicotine and its metabolite, cotinine, in the rat as a function of time. *Pharmacology* 50:128-36

197. Save E, Poucet B. 2000. Involvement of the hippocampus and associative parietal cortex in the use of proximal and distal landmarks for navigation. *Behavioural Brain Research* 109:195-206

198. Schoenmakers T, Wiers RW, Jones BT, Bruce G, Jansen A. 2007. Attentional re‐training decreases attentional bias in heavy drinkers without generalization. *Addiction* 102:399-405

199. Schoenmakers TM, de Bruin M, Lux IF, Goertz AG, Van Kerkhof DH, Wiers RW. 2010. Clinical effectiveness of attentional bias modification training in abstinent alcoholic patients. *Drug and Alcohol Dependence* 109:30-6

200. Schooler C, Feighery E, Flora JA. 1996. Seventh graders' self-reported exposure to cigarette marketing and its relationship to their smoking behavior. *American Journal of Public Health* 86:1216-21

201. Shadel WG, Martino SC, Setodji C, Scharf D. 2012. Momentary effects of exposure to prosmoking media on college students' future smoking risk. *Health Psychology* 31:460

202. Shaham Y, Miczek KA. 2003. Reinstatement—toward a model of relapse. *Psychopharmacology* 168:1-2

203. Shiffman S. 2009. Ecological momentary assessment (EMA) in studies of substance use. *Psychological Assessment* 21:486

204. Shiffman S, Engberg JB, Paty JA, Perz WG, Gnys M, et al. 1997. A day at a time: predicting smoking lapse from daily urge. *Journal of Abnormal Psychology* 106:104

205. Shiffman S, Patten C, Gwaltney C, Paty J, Gnys M, et al. 2006. Natural history of nicotine withdrawal. *Addiction* 101:1822-32

206. Shiffman S, Paty JA, Gnys M, Kassel JA, Hickcox M. 1996. First lapses to smoking: within-subjects analysis of real-time reports. *Journal of Consulting and Clinical Psychology* 64:366

207. Shiffrin RM, Schneider W. 1977. Controlled and automatic human information processing: II. Perceptual learning, automatic attending and a general theory. *Psychological Review* 84:127

208. Siegel R, Ward E, Brawley O, Jemal A. 2011. Cancer statistics, 2011. *CA: A Cancer Journal for Clinicians* 61:212-36

209. Silagy C, Lancaster T, Stead L, Mant D, Fowler G. 2004. Nicotine replacement therapy for smoking cessation. *The Cochrane Library*

210. Slemmer JE, Martin BR, Damaj MI. 2000. Bupropion is a nicotinic antagonist. *Journal of Pharmacology and Experimental Therapeutics* 295:321-7

211. Solar O, Irwin A. 2007. A conceptual framework for action on the social determinants of health. World Health Organization: Commission on Social Determinants of Health: Geneva.

212. Solomon RL. 1980. The Opponent-Process Theory of Acquired Motivation «unci. *American Psychologist*:691

213. Sorlie P, Rogot E, Anderson R, Johnson NJ, Backlund E. 1992. Black-white mortality differences by family income. *The Lancet* 340:346-50

214. Stead LF, Lancaster T. 2002. Group behaviour therapy programmes for smoking cessation. *The Cochrane Library*

215. Stead LF, Perera R, Bullen C, Mant D, Lancaster T. 2008. Nicotine replacement therapy for smoking cessation. *The Cochrane Library*

216. Stead LF, Perera R, Lancaster T. 2007. A systematic review of interventions for smokers who contact quitlines. *Tobacco Control* 16:i3-i8

217. Stewart J, De Wit H, Eikelboom R. 1984. Role of unconditioned and conditioned drug effects in the self-administration of opiates and stimulants. *Psychological Review* 91:251

218. Stone AA, Shiffman S, Schwartz JE, Broderick JE, Hufford MR. 2002. Patient non-compliance with paper diaries. *BMJ* 324:1193-4

219. Tiffany ST. 1990. A cognitive model of drug urges and drug-use behavior: role of automatic and nonautomatic processes. *Psychological Review* 97:147

220. Tønnesen P, Tonstad S, Hjalmarson A, Lebargy F, Van Spiegel P, et al. 2003. A multicentre, randomized, double‐blind, placebo‐controlled, 1‐year study of bupropion SR for smoking cessation. *Journal of Internal medicine* 254:184-92

221. Trinidad DR, Gilpin EA, White MM, Pierce JP. 2005. Why does adult African-American smoking prevalence in California remain higher than for non-Hispanic whites? *Ethnicity and Disease* 15:505

222. Trinidad DR, Pérez-Stable EJ, White MM, Emery SL, Messer K. 2011. A nationwide analysis of US racial/ethnic disparities in smoking behaviors, smoking cessation, and cessation-related factors. *American Journal of Public Health* 101:699-706

223. U.S. Department of Health and Human Services CfDCaP, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health. 1998. Tobacco Use Among U.S. Racial/Ethnic Minority Groups—African Americans, American Indians and Alaska Natives, Asian Americans and Pacific Islanders, and Hispanics: A Report of the Surgeon Genera. ed. CfDCaP U.S. Department of Health and Human Services, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health. Atlanta

224. Unger JB, Cruz TB, Schuster D, Flora JA, Johnson CA. 2001. Measuring exposure to pro-and anti-tobacco marketing among adolescents: intercorrelations among measures and associations with smoking status. *Journal of Health Communication* 6:11-29

225. United States Department of Health and Human Services. 1988. The health consequences of smoking: Nicotine addiction. A Report of the Surgeon General U.S. Government Printing Office.

226. Wakefield M, Germain D, Henriksen L. 2008. The effect of retail cigarette pack displays on impulse purchase. *Addiction* 103:322-8

227. Waters AJ, Li Y. 2008. Evaluating the utility of administering a reaction time task in an ecological momentary assessment study. *Psychopharmacology* 197:25-35

228. Waters AJ, Marhe R, Franken IH. 2012. Attentional bias to drug cues is elevated before and during temptations to use heroin and cocaine. *Psychopharmacology* 219:909-21

229. Waters AJ, Sayette MA. 2006. Implicit cognition and tobacco addiction. *Handbook of implicit Cognition and Addiction*:309-38

230. Waters AJ, Shiffman S, Sayette MA, Paty JA, Gwaltney CJ, Balabanis MH. 2003. Attentional bias predicts outcome in smoking cessation. *Health Psychology* 22:378

231. Waters AJ, Shiffman S, Sayette MA, Paty JA, Gwaltney CJ, Balabanis MH. 2004. Cue-provoked craving and nicotine replacement therapy in smoking cessation. *Journal of Consulting and Clinical Psychology* 72:1136

232. Waters AJ, Szeto EH, Wetter DW, Cinciripini PM, Robinson JD, Li Y. 2014. Cognition and craving during smoking cessation: an ecological momentary assessment study. *Nicotine & Tobacco Research* 16:S111-S8

233. Webb MS. 2008. Treating tobacco dependence among African Americans: A meta-analytic review. *Health Psychology* 27:S271

234. Weiss JW, Cen S, Schuster DV, Unger JB, Johnson CA, et al. 2006. Longitudinal effects of pro-tobacco and anti-tobacco messages on adolescent smoking susceptibility. *Nicotine & Tobacco Research* 8:455-65

235. West R, Baker CL, Cappelleri JC, Bushmakin AG. 2008. Effect of varenicline and bupropion SR on craving, nicotine withdrawal symptoms, and rewarding effects of smoking during a quit attempt. *Psychopharmacology* 197:371-7

236. Whitehead M, Dahlgren G. 2006. Concepts and principles for tackling social inequities in health: Levelling up Part 1. *World Health Organization: Studies on social and economic determinants of population health* 2

237. WHO Commission on Social Determinants of Health World Health Organization. 2008. *Closing the Gap in a Generation: Health Equity Through Action on the Social Determinants of Health: Commission on Social Determinants of Health Final Report*. World Health Organization

238. Williams DR, Collins C. 2001. Racial residential segregation: a fundamental cause of racial disparities in health. *Public Health Reports* 116:404

239. Wise RA, Bozarth MA. 1987. A psychomotor stimulant theory of addiction. *Psychological Review* 94:469

240. Wise RA, Koob GF. 2014. The development and maintenance of drug addiction. *Neuropsychopharmacology* 39:254-62

241. Wood AJ, Mendelson JH, Mello NK. 1996. Management of cocaine abuse and dependence. *New England Journal of Medicine* 334:965-72

242. Yerger VB, Wertz M, McGruder C, Froelicher ES, Malone RE. 2008. Nicotine replacement therapy: Perceptions of African-American smokers seeking to quit. *Journal of the National Medical Association* 100:230-6